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CONGENITAL BILATERAL ABSENCE OF KIDNEYS IN A 140-MILLIMETER PIG EMBRYO[1]

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INTRODUCTION

ONE PLATE AND 3 TEXT FIGURES

A review of the literature on renal malformations and maldevelopments has demonstrated the fact that, while unilateral congenital absence and maldevelopment of kidneys have been frequently recorded in the human and lower animals, bilateral cases, though observed with comparative frequency in embryos, have been reported but once or twice in older human beings.

There is naturally an obvious reason for this difference in frequency of occurrence. While unilateral congenital absence or defective conditions of kidneys are compatible with extrauterine life, when both organs are wanting, life is hardly conceivable; and, of course, cases that go on to adult life are much more apt to be observed and reported than those conditions which might exist only in utero. It is also for this reason that congenital unilateral renal defects acquire much greater importance to the clinician and to the surgeon than similar bilateral defects. It is now a surgical routine for men doing kidney work to keep in mind congenital unilateral absence of kidney. Nephrectomy in such cases would invariably be fatal.

As to absence of both kidneys, however, though it lacks the clinical importance of unilateral renal aplasia or congenital absence of one kidney, its developmental significance is of such interest to students of embryology that a careful report of all such cases would seem warranted. It is for this account that

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the present case is reported, especially since the rest of the urogenital tract seemed to be apparently normal, differing in this respect from many of the cases so far recorded.

Whereas up to date some three hundred cases of unilateral absence of kidneys have been reported, a careful search of the literature on hand shows only the following men reporting instances of bilateral renal absences.

Gerard(4) cites Moulon, Beclard, Meyer de Bonn, Chausier, Pignée, and Dubierre as having reported cases of congenital absence of both kidneys. In the case of Moulon, a girl about fourteen years of age, both kidneys and ureters were absent, while the umbilicus opened in the region of the mons veneris and constantly exuded a liqüid resembling urine. Ahlfeld,(1) English,(3) Hauch,(6) Rud Bayer,(7) and Zaufal(9) also reported cases of bilateral renal absence. Greenfield(5) describes two cases of a similar defect, in one of which the vas depreus and the testicles also were absent. Schaffaer(8) mentions the case of a 7-month-old human fœtus without any traces of kidneys, and Coen,(2) according to Ballowitz, in a study of thirty-three monstrosities, found fifteen cases of total absence of kidneys, in which the adrenals were larger than normal.

There are probably more cases reported than those mentioned, but with the literature available it has been impossible for us to add any more or even to verify some of the above reports.

MATERIAL USED

The present specimen was obtained while on a trip to the slaughterhouse of the city to inject kidneys of pig embryos. The case is a female pig embryo, 140 millimeters long, from a litter of five apparently perfectly normal specimens of approximately equal size.

ANATOMY AND HISTOLOGY

In external appearance and on careful examination of the thoracic and abdominal cavities, this specimen appears normal. Our attention was called to the absence of the kidneys only after looking in vain for them to make our injection. The genitalia also appear normal; the ovaries measure 5.5 by 3 by 3 millimeters. The oviducts and uterus are in every respect identical with those of pigs of the same size.

In the urinary apparatus the bladder is well developed, measuring 12 by 4 by 4 millimeters and ending in a well-formed urachus. The two hypogastric arteries are also well defined and normally located. The kidneys are absent, and no traces of what might be taken for rudiments of these organs can be found in the neighborhood of their normal position. The two ureters are well developed, and their orifices at the trigone are patent. The right ureter, which appears slightly larger and longer than the left, begins from the right inferolateral side of the bladder, passes in an upward and lateral direction for about 9 millimeters, then continues upward in a wavy course over the quadratus lumborum for about 16 millimeters, and ends in a funnel-shaped expansion which is lost in the retroperitoneal fascia in this region. The left ureter, slightly smaller in diameter than the right, is decidedly shorter and lacks an abdominal portion. Traced from the lower inner side of the bladder, it passes in a lateral direction to the back of the pelvis with a slight concavity upward for a distance of 6.5 millimeters and ends in a thin membrane attached to the posterior pelvic wall.

Occupying apparently normal positions, the adrenals appear as two large, more or less pyramidal masses which are about three times as large as those of pig embryos of approximately the same size.

Retroperitoneally, over the right quadratus lumborum, and on a level with and lateral to the expanded upper end of the right ureter, a small mass of glandularlike tissue of irregular shape is found. This was removed, marked "G," and imbedded for histological examination. Two similar bodies were seen on the right lateral portion of the posterior abdominal wall, one immediately above and the other to the mesial side of the right ovary; both were removed for embedding and marked "D" and "H," respectively. On the left side two similar masses were also seen, one of triangular shape a few millimeters lateral to the left ovary, and the other oval in shape, immediately below it. These were embedded as "B" and "A" respectively. No other masses of tissue which might be looked upon as suspicious traces of kidney were seen in this region (fig. 1).

The abdomens of other pig embryos approximately of the same size were opened and studied for comparison.

In a pig embryo 120 millimeters in length the right kidney measured 15 by 7.5 by 7 millimeters; and the left, 15.5 by 7.5 by 6.5 millimeters. Both were situated on the posterolateral wall of the abdomen and were so prominent as to be the first things noticed on lifting up the coils of the intestine. Capping both organs were the two triangular adrenals which were about one third as large as those found in our kidneyless specimen. The right ureter was about as large in diameter as the corresponding ureter of the pig in question. It started from the pelvis of the kidney and followed a similar course down to the bladder. The left ureter was also well developed; it began from the pelvis of the left kidney, passed downward parallel with the hypogastric artery for about 15 millimeters behind the broad ligament of the uterus, and then made a turn medially for about 5 millimeters and buried itself in the lower pole of the bladder. The ovaries were both normally placed, and the right measured 4 by 2.75 by 2 millimeters. The rest of the abdominal organs appeared normal and corresponded identically with those in the specimen.

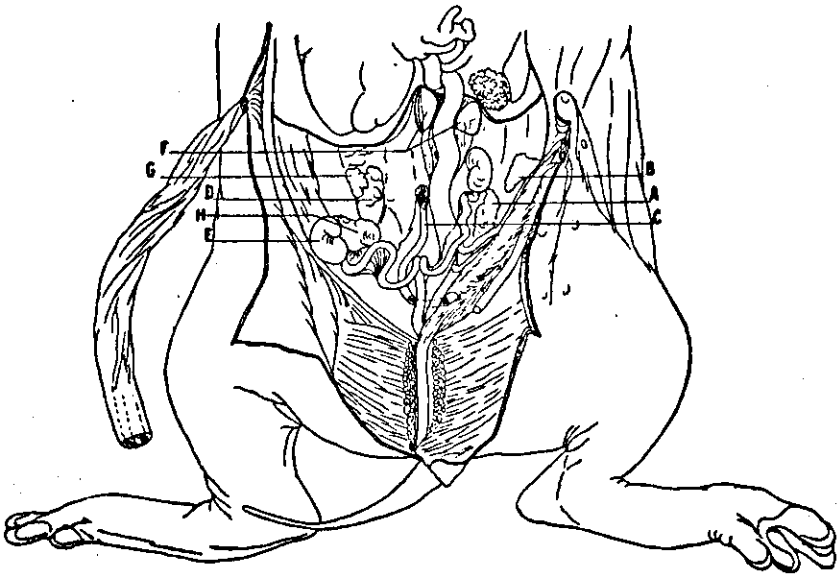

FIG. 1. Schematic drawing of abdominal cavity with intestinal coils reflected. × 3.

Owing probably to prolonged fixation in formalin there was a generalized contraction of the tissues of our specimen. All the sections were stained with Delafield's hæmatoxylin and eosin.

Sections from the right ovary showed a richly vascular medulla and a cortex studded irregularly with primitive follicles, many of which contained a well-formed ovum. Between these follicles, and embedded in a reticulum of young connective tissue, cords of Pflüger were plainly seen. The stroma of both the medulla and the cortex was composed of young connective tissue cells covered by a germinal epithelium which was torn in many places. The outer end of the right oviduct was also sectioned and stained. It showed a well-formed lumen lined with pseudostratified, columnar, ciliated epithelium surrounded by a layer of vascular, young connective tissue in many places richly infiltrated by lymphocytes. Between the section of the oviduct and the rete ovarii, in the mass of tissue which represents the broad ligament, several distinct mesonephric tubules, which are part of the parovarium, were seen.

The last 6 millimeters of the upper end of the right ureter was embedded for examination. Sections from the lower end of this piece of ureter showed a well-defined oval lumen lined with transitional epithelium, the cells of which for the most part were ovoid in shape. Their nuclei stained faintly and were placed near the cell base. Surrounding this epithelium, two distinct layers of tissue were seen; the inner coat, slightly the thicker of the two, appeared to be made up of young connective tissue cells which were of irregular shape and showed collections of lymphoid tissue in many places. The outer layer was made up of fusiform cells more loosely arranged. In this coat numerous blood vessels were seen. Sections taken higher up had smaller lumen lined with transitional epithelium, the cells of which appeared better stained and their outlines more distinct. The surrounding connective tissue layers were looser in texture, the cells becoming more starshaped and approaching more the character of mesenchymal cells. Higher up the ureter breaks up into two or three solid cords of epithelioid cells surrounded by loose mesenchymatous tissue, which now exhibits here and there collections of lymphoidlike cells resembling to a certain extent small lymph glands. In no section of the entire 6 millimeters of ureter could we find any tissues which might in any way suggest the slightest structure of the blastemal cap, which is always present in normal cases (figs. 2 and 3).

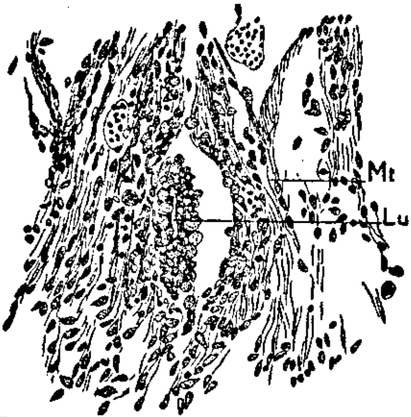

FIG. 2. Section of upper end of ureter (right).

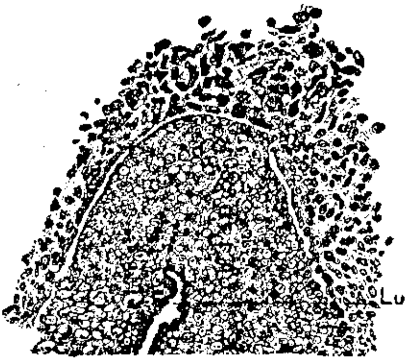

FIG. 3. Section of lower end of right ureter.

Sections from both adrenals showed a well-defined capsule of white fibrous connective tissue. Between this capsule and the glandular substance is a narrow strip of vascularized young connective tissue, in which irregularly shaped masses of cortical and medullary cells were to be seen. The cortex of the gland appeared as a narrow band of cellular elements surrounding the medulla. This cortex is composed of a vascular reticulum in the meshes of which cortical cells, polygonal in shape, are arranged in vertical cords distinctly revealing the beginning of of the zonas glomerulosa and fasciculata. Toward the periphery these were granular and showed the characteristic fat granules observed in adult cells; their nuclei, ovoid and round, filled from one fourth to one half of the cell body and for the most part were centrally located. Toward the center these cells gradually merged with those of the medulla. The medulla, comparatively more abundant than in adult adrenals, was separated at places from the cortex by a large blood vessel. There is a distinct vascular reticulum, the mesh spaces of which were filled with irregularly shaped medullary cells, with large ovoid nuclei placed centrally and almost filling the entire body of the cells. These cellular elements were arranged in irregular branching masses not unlike those of the parathyroid glands. The blood vessels were for the most part thin-walled with a distinct endothelial lining.

Sections from masses "A," "B," "G," and "H" were apparently identical. There was a thin fibrous connective tissue capsule with adipose deposits at places, within which was a delicate, vascularized connective tissue reticulum. The mesh spaces of this reticulum were lined and contained large flattened and irregularly ovoid cells with round nuclei centrally placed and filling about one fourth or one half of the body of the cells. Masses and isolated cells of lymphoid nature were to be seen. These cellular elements were seen to be separated from the lining of the sinuses and from the reticulum by narrow spaces. In some sections, especially in those of "H," small eosinophilic cells were also observed. These cellular bodies were small lymph glands.

Sections from "D" were apparently made up of vascularized embryonic connective tissue.

EMBRYOLOGY

In considering the embryology of the urinary excretory system, especially in amniotes, we are at once impressed with the fact that we are dealing not with a gradual development of one single organ which is laid down and gradually brought up to its definitive completion, but to a saltatory development of three different organs, the pronephros, the mesonephros, and the metanephros, which are developed and formed in succession, each being apportioned and adapted to a definite period of one entire development. Of these organs the pronephros and the mesonephros are merely provisional kidneys; their activities become superfluous and partially or totally disappear and are eventually supplanted by the last and third organ, the metanephros, which becomes the adult organ of urinary excretion.

The anlage of these three organs is the same; the cells of the intermediate mass of mesoderm of the embryo, from the cephalic portion of which develops the pronephros, from the middle zone the mesonephros, and from the caudal end the metanephros.

In the present case the pronephros has evidently undergone its full development and degeneration, leaving only behind its primary collecting duct from which the two ureters have apparently normally developed.

That the mesonephros has also undergone its normal course of development and degeneration is evidenced by the presence of distinct mesonephric tubules in the region of the rete ovarii forming the parovarium, the normally formed uterus and oviduct, and the two well-developed ureters, which are all derivatives of the mesonephros.

In the case of the metanephros, the ureter in the right side has apparently developed upward to its normal length, and its upper extremity has started to expand into a funnel-shaped primitive pelvis and to divide into several primary collecting tubules. No evidences can be seen, however, of a blastemal cap accompanying it in this upward growth. The left ureter has apparently become arrested at the brim of the pelvis, becoming entangled there in the retroperitoneal mesenchyma.

Whether these ureters were accompanied by blastemal tissue from the nephrogenic cord which might have undergone complete regression or not is a question which cannot be fully determined with the present findings.

SUMMARY AND CONCLUSIONS

The review of the literature has shown the comparative rarity of congenital absence of kidneys, especially in man. Only a few cases have so far been reported, one or two of which are inconceivable.

The absence of the two kidneys, though embryologically interesting, lacks the clinical importance of the unilateral cases, owing to the fact that it is incompatible with life.

The specimen studied in the present case was an apparently normally developed embryo in no outward point different from the others of the same litter.

There are apparently no other abnormalities found in the rest of the urinary apparatus aside from the absence of both kidneys and the short and maldeveloped left ureter. Both ureters were normally placed, and both ended in expanded extremities, which were lost in the surrounding fascia.

The genital tract appeared normal as compared with embryos of approximately the same size.

The adrenals were larger than normal and appeared rather advanced in differentiation.

There were no traces of tissues in the region of the nephrogenic cord, which on microscopic study might suggest the least appearance of renal blastema. The four or five glandular collections, which might be regarded as suspicious, were demonstrated to be lymphoid bodies and masses of mesenchymatous tissue without signs of tubular formation whatsoever.

From the present case the following conclusions may be drawn:

1. In the case of the right ureter the absence of kidney is probably due to a failure of the nephrogenic blastemal cap to appear or to develop, the upper expanded end of this ureter having undergone tubular divisions preparatory to the formation of the medulla of the kidney.
2. The left ureter may have been arrested in its growth cephalad and become entangled with the mesenchyma of the pelvis, thus failing to reach the nephrogenic cord and meeting the blastemal substance which might have existed and undergone regression.
3. From the apparently normal development in general of this specimen as compared with the others of the same litter it is probable that kidneys are not essential for proper growth intra utero.
4. The adrenals said to be generally larger in renal absence as reported by Coen and others is also confirmed in this specimen.

This work has been done under the immediate supervision of Dr. E. S. Ruth, to whom I wish to express my sincere thanks for kind and very helpful suggestions. I also wish to thank Dr. B. C. Crowell for valuable help in the microscopical study of the sections.

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ILLUSTRATIONS

[From drawings by Juan Castro.]

PLATE I. Female pig embryo, 14 centimeters long. ×3.

TEXT FIGURES

FIG. 1. Schematic drawing of abdominal cavity with intestinal coils reflected. ×3. *A, B, G, H*=lymphoid collections; *F*=adrenals; *D*=mesenchymatous body; *E*=right ovary; *C*=upper end of right ureter.

2. Section of upper end of ureter (right). *Lu*=lumen of ureter; *Mt*=mesenchymatous tissue. Objective 6; occular 4, Leitz.

3. Section of lower end of right ureter. *Lu*=lumen of ureter. Objective b; occular 4, Leitz.

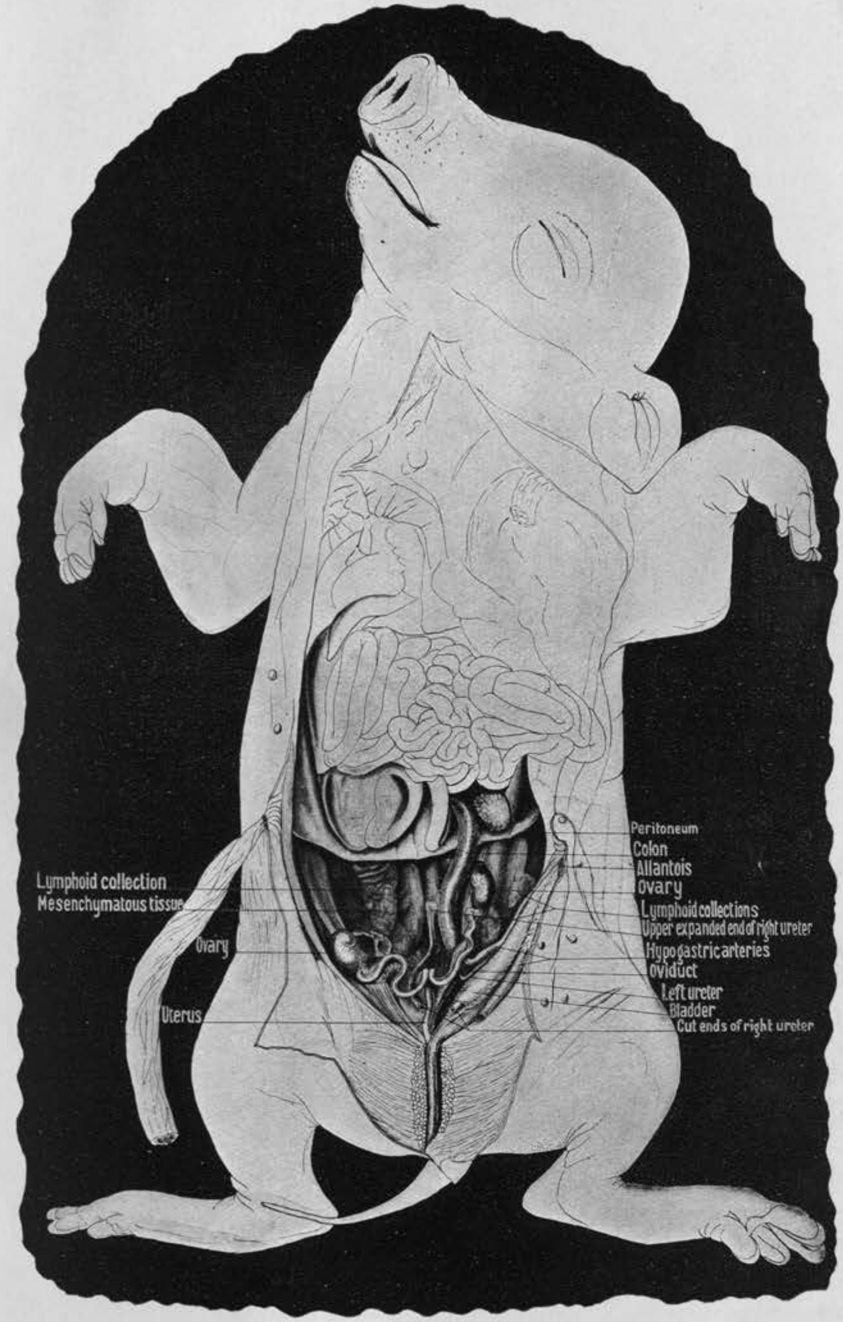

PLATE I. FEMALE PIG EMBRYO, 14 CENTIMETERS LONG.

POISONING BY ILLICIUM RELIGIOSUM SIEBOLD[1]

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Poisoning from the use of parts of plants does not occur very frequently in the Philippine Islands. We are not aware of any official statistics or publication on this subject; neither are we aware that the various species of poisonous plants which abound in the Philippine flora have been defined and enumerated. But in reality poisoning from the use of these plants in the Islands is by no means as rare as one would be led to believe after consulting the literature on the subject. We know from reliable sources that in Manila and in the provinces have occurred intentional and accidental cases of poisoning by *namí* (*Dioscorea daemona*), *maná* (*Jatropha multifida*), *suma* (*Anamirta cocculus*), *salagó* (*Wikstroemia ovata*), *katbalonga* (*Strychnos ignatii*), *talampunay* (*Datura alba*), *tuba* (*Jatropha curcus*), and other plants of still undetermined status.

REPORT OF FOUR CASES OF POISONING BY ILLICIUM RELIGIOSUM SIEBOLD WHICH OCCURRED IN MANILA

Recently we have had occasion to observe four cases of poisoning from the use of a decoction of *sanki*, the fruit of *Illicium religiosum* Siebold. Since the symptomatology and therapy of this poisoning are not well defined, and no mention of them is made in the textbooks of tropical medicine in current use except that by Grall and Clarac(1) and by Mense,(2) the report of these cases might be interesting and of some clinical value.

CASE 1

Mr. N. N., 50 years old, believed through hearsay that a decoction of sanki would be a good stimulant, stomachic, and excellent remedy for cholera. On October 24, 1914, at 3 o'clock in the afternoon, he took two cups of a strong decoction of sanki (exact quantity of the poison taken undetermined). At 4 o'clock, that is, one hour later, repeated vomitings occurred. These were accompanied by bowel evacuation of large quantities of fluids, cramp in the calf muscles, profuse perspiration, and intense thirst. The patient complained of severe headache, vertigo, and extreme weakness, especially in the lower extremities. At 4.30 he became restless, tossed in bed, and after a

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few minutes was seized by clonic and tonic convulsions. During the attack of convulsions the face was congested and expressed fear, the respiration stopped, the eyeballs diverged, the head was retracted toward the back, and the trunk and the extremities were rigid and extended, giving now and then slight convulsive movements. After this single attack the patient was exhausted and markedly prostrated, but was conscious and showed no sign of disturbance of mental equilibrium. The pulse was large and strong at the beginning, but became rapid and thready afterward; there was dyspnœa, and the urine was scanty and highly colored. The presence of albuminuria was not determined.

With the belief that we were dealing with a case of cholera, the patient was given 500 cubic centimeters of physiological salt solution and 1 cubic centimeter of camphorated oil, injected hypodermically, and hot stimulating drinks. Five hours after the appearance of the first symptom the patient was distinctively improved and rapidly recovered.

CASES 2 AND 3

These cases are the daughter and the cook of Mr. N. N. (case 1), each of whom took a very small quantity of a decoction of sanki on the advice of the latter. Emesis appeared shortly after taking the poison, and the patients complained of no disturbances other than temporary dizziness. The mildness of the symptoms in these two cases was undoubtedly due to the early onset of vomiting and presumably to the small quantity of the poison ingested.

CASE 4

This case was a woman who three days after labor was given by her mother a cup of sanki decoction as a stimulant. Again the exact quantity of the poison ingested unfortunately could not be determined. Four hours after taking the poison the patient developed the following symptoms: Vomiting, cephalalgia, cramps in the legs, convulsions resembling those in case 1, profuse sweating, and oliguria. Diarrhœa, restlessness except during the convulsions, and marked prostration did not occur. The administration of chloral hydrate caused the convulsions to disappear; the patient recovered.

Dr. Tee Han Kee has very kindly communicated to us privately that more than twenty cases of poisoning by *Illicium religiosum* have come under his observation. These cases were all Chinese residing in Manila, who used the poison in committing suicide or for the purpose of relieving tympanites and various ailments, such as hydrocele and anasarca, which they attribute to cold.

He has also kindly sent to us a detailed description of three more cases in Chinese girls recently attended by him. All the girls developed convulsions, and one of them died.

ETIOLOGY OF POISONING BY ILLICIUM RELIGIOSUM

The fruit of *Illicium religiosum* is not used as a condiment nor as a medicine. Under the name of *Illicium anisatum* it is employed in some towns of the Orient in small, indefinite quantities to flavor certain native hodgepodges. The Chinese in Manila acknowledge that the fruit of this plant is highly poisonous; yet they do not distinguish it from *Illicium anisatum*, and they call indiscriminately these two species of *Illicium* fruits *pue-kag* (star of eight rays). They consider both species poisonous. Eykman(3) and Langaard(4) established experimentally the toxicity of *Illicium religiosum*, but so far as we know the relative toxicity of *Illicium anisatum* and *Illicium religiosum* is not yet determined. According to our unpublished experiments carried on in the department of pharmacology, College of Medicine and Surgery, University of the Philippines, in collaboration with Miss Felisa Nicolas, the fruit of *Illicium religiosum* is fourteen times more toxic than that of *Illicium anisatum* when kittens are used as the test objects. The fruit of *Illicium religiosum*, in the proportion of 0.25 gram per kilogram of body weight, was injected hypodermically in the form of an infusion. It is a sure minimal fatal dose to these animals. *Illicium anisatum*, on the other hand, is extensively used by the laity as a stimulant, stomachic, and carminative. The Filipinos use it in the preparation of various dishes and to flavor a drink known as *tahu* or *salabat*, which is nothing more than a sweetened decoction of ginger. Tahu is popular in many provinces of Luzon, and the poor people drink it in place of tea and coffee. At the present time *Illicium religiosum* is the only variety which is sold in the groceries of Manila, while *Illicium anisatum* is obtainable only from the drug stores. It is, therefore, not strange that cases of poisoning by *Illicium religiosum* should occur in Manila. It is highly probable that it is the materies morbi of the so-called *colerinas* which have been observed to follow the ingestion of salabat flavored with sanki in some towns of the Philippines. On account of the cheapness of *Illicium religiosum*, it is frequently substituted for *Illicium anisatum*. This substitution has been a cause of the poisoning recorded in Holland, in France, and in other countries and is illustrated by the cases observed by Montel (5) in three Europeans in Conchin China. These cases present different stages of the poisoning, and we consider it interesting to reproduce them in detail.

CASE 1

Mr. O. had been taking infusions of *Illicium anisatum* for diarrhœa. Having used his provision, he ordered more *Illicium anisatum* from a drug store, on October 6, but his servant went to a Hindu grocery and obtained *Illicium religiosum.* Mr. O. took of it in the form of an infusion about 7.30 of the same morning. At 9 o'clock he began to have repeated vomitings, profuse diarrhœa, and cramps in the legs.

Convulsions set in at 11 o'clock. The patient became suddenly restless and unconscious after giving a loud cry. During the convulsion the breathing stopped, the eyeballs rotated laterally, the face expressed fear, the muscles of the trunk contracted, the head was retracted toward the back, the forearms were flexed, the hands were clinched, and the limbs were rigid, giving occasional jerks. This paroxysm lasted one or two minutes and resembled that of the classical idiopathic epilepsy. After ten minutes consciousness suddenly returned, the respiration was quite, and the patient complained of cramps in the legs.

Between 11 o'clock in the forenoon and 9 o'clock in the evening this convulsive paroxysm was repeated five times. The patient, at the interparoxysmal periods, was exhausted, complained of severe headache, was restless in bed, and assumed a Z-position. He was thirsty and drank a large quantity of Vichy water, which he vomited. The vomiting was frequent; it lasted till 3 o'clock in the afternoon. There were anuria, carphology, and clouding of consciousness; the patient was not able to recognize the persons surrounding him. The pulse was 120 beats per minute and was small.

The patient was given 0.5 gram of caffeine, 1 cubic centimeter of camphorated oil injected hypodermically, and a little antipyrine which seemed to alleviate the headache. He was also injected with physiological salt solution, 500 cubic centimeters at noon and another 500 cubic centimeters at 10 o'clock in the evening. After the second injection the general condition was improved and the convulsions did not return. At night he passed urine and slept.

On the morning of October 7 he urinated again and slept a little. The headache disappeared, but there were still mental dullness and physical weakness. In the afternoon the temperature was 38.7° C., the pulse was 106 beats per minute, and there were asthenia and somnolence. The temperature rose to 40° C. at 10 o'clock in the evening, but later it subsided. The patient urinated seven times during the night.

On October 8 the general condition of the patient was excellent. There was only slight mental depression, which gradually disappeared. The patient recovered completely and had no recollection of anything that had happened after the ingestion of the poison. He did not remember the approximate quantity of the *Illicium religiosum* which he had taken.

Cases 2 and 3 were Mr. and Mrs. B., who one afternoon took an infusion of *Illicium religiosum*. Mrs. B. drank a smaller portion of the poison. She had early vomiting and profuse diarrhœa. Vomiting did not appear in Mr. B. until one hour later, and the vomitus was less abundant. They developed convulsions resembling those described in the case of Mr. O. Mrs. B. recovered, but her husband, in addition to the convulsions, suffered from anuria, hallucination with tendency to commit violence, and marked paresis of the lower limbs. On the third day he showed considerable improvement, but afterward, anuria, restlessness, insomnia alternating with depression, cyanosis, cold extremities, and œdema in the calf muscles manifested themselves, and on the eighth day the patient died of asphyxia. Post-mortem examination revealed congestion of the alimentary tract and enlarged, congested kidneys.

Geerts(6) reported six cases of poisoning by *Illicium religiosum*. These cases occurred in Kanagawa, near Yokohama, Japan, and resulted from the substitution of the sesame oil by the fixed oil of *Illicium religiosum* in the preparation of food. In one of these cases death occurred. Other cases of poisoning arising from the substitution of *Illicium religiosum* for *Illicium anisatum* have also been reported by Planchon and Dreyer,(7) Vogl,(8) Barral,(9) Delotte,(10) and Lamarque.(11)

In addition to these cases Eykman mentioned some cases of poisoning which took place at Leeuwarden in the Netherlands, which were caused by fruits which corresponded in botanical description and pharmacological action to the fruits of *Illicium religiosum*. He further mentioned five cases of poisoning in five children at Uyeno Park, Tokyo. These children were found playing at the park, foaming at the mouth and violently convulsed. The opinion that these were cases of poisoning by *Illicium religiosum* was based upon the occurrence of a number of fruit-bearing specimens of the plant in Uyeno Park in the neighborhood of the place where the children were playing and the discovery of some seeds of *Illicium religiosum* in the sleeves of the dresses, in the vomitus of one child, and in the fæces of another. Three of the children died. There were observed contracted pupil in one, mydriasis and bloody vomitus in three cases, and foaming at the mouth, which was a prominent symptom in our experiments on cats. Otherwise the symptomatology was, in general, similar to that described by Montel and by us.

SYMPTOMATOLOGY AND DIAGNOSIS OF POISONING BY ILLICIUM RELIGIOSUM

According to these cases the following symptoms may occur in cases of poisoning by *Illicium religiosum:* Foaming at the mouth, repeated vomiting, diarrhœa, thirst, unconsciousness, clonic and tonic convulsions, cramps in the legs, cephalalgia, mental disturbances, insomnia, profuse sweating, oliguria or anuria, large strong pulse giving place to small rapid pulse, cold extremities, contracted or dilated pupils, paresis of the lower limbs, and exhaustion. The vomitus may be streaked with blood. During the convulsions the pupils as a rule dilate, the eyeballs bulge, the head is retracted toward the back, the respiration stops, and the face is cyanotic. According to Burdin (12) paresis of the limbs was the most prominent symptom in all the cases which came under his observation. Ferraud (13) claims that it can be reproduced exclusively by varying the dose of *Illicium religiosum* given to experimental animals. This we have not yet studied carefully. In severe or fatal cases of poisoning in man, repeated vomiting, diarrhœa, clonic and tonic convulsions which appear in paroxysm, cramps in the legs, and retraction of the head appear to be the most conspicuous symptoms. Cholera, strychnine poisoning, tetanus, and perhaps cerebrospinal meningitis may be confounded with *Illicium religiosum* poisoning; but the history that the above symptoms followed the ingestion of the *Illicium*, and its presence in and about the house and particularly in the vomitus and bowel evacuation of the patient, should readily clear the difficulty in establishing a definite diagnosis.

IDENTIFICATION OF ILLICIUM RELIGIOSUM

The fruit of *Illicium religiosum*, according to G. Planchon and E. Collin,(14) is generally two thirds the size of that of *Illicium anisatum*. The former seldom develops the full number of carpels, and for this reason the rays formed by them are rarely regular. The superior border of the carpel is never a horizontal line, but it turns upward at the end, giving the individual ray the form of a claw. The depression on the lateral surface produced by the adjacent carpel is generally conical in outline, while in *Illicium anisatum* it is semiellipsoid. The seed of *Illicium religiosum* is a trifle smaller and presents an obtuse apex, due to the development of the raphe. When the fruit is powdered, it gives off the odor of cubeb or laurel and not the unmistakable anis odor of *Illicium anisatum.* Vogl (15) states that the sclerenchyma layer lining the endocarp at the ventral suture is softer and is composed of cells whose walls are in general thinner than in *Illicium anisatum.* He also states that the cells of the endocarp at this level are shorter than in the last species. The content of these cells turns brownish black on coming in contact with a hot solution of potassium hydroxide. Godfrin (16) emphasizes the fact that the differential characteristics are to be sought for in the seeds. He claims that the seed of *Illicium anisatum* contains only parenchymatous cells under the external sclerenchyma layer of its envelope, while *Illicium religiosum* contains sclerenchyma cells in the corresponding part of the seed. Rud. Pfister (17) bases the differentiation on the form and size of the grains of the aleurone contained in the seed. They are generally ovoid, and the globules contained in them are very small and appear like rounded dots in *Illicium religiosum;* they are larger and irregularly sinuous or tuberous in *Illicium anisatum.* Holmes (18) claims that the two species of *Illicium* react differently to blue litmus paper; *Illicium religiosum* when moistened turns it bright red, *Illicium anisatum* only pale red. Montel summarizes in the following table the distinguishing characters of the two species:

TABLE I.—*Differential characters of the fruits of Illicium religiosum and of Illicium anisatum.*

Differential characters.	*Illicium religiosum.*	*Illicium anisatum.*
Odor	Suggests the odor of pepper and of yew-tree.	Of anise.
Seed	Slightly ovoid	Flattened.
Follicles	Slender, the surface rough, the ends pointed, curved in the form of a claw.	Stout, woody, the surface less rough, ends obtuse.
Axis	Tapers gradually upward, terminates slightly below the extremity of the borders of carpels, and appears to incline somewhat toward the lower part of the latter.	Slightly truncated above, forming a disc in to which the fruits are inserted.
Sclerenchyma cells of the peduncle and of the axis.	Few, small, rounded or elliptical.	Numerous, large, and their wall thick.
Anetol	Absent	Considerable.

According to Montel the easiest method of differentiating one *Illicium* from the other is to boil a follicle of the fruit with one or two cubic centimeters of alcohol in a test tube. With *Illicium anisatum* the addition of water to the alcoholic solution causes the formation of an opalescent precipitate resembling that of absinth of commerce, due to the precipitation of anetol. With *Illicium religiosum* the alcoholic solution remains limpid, but on evaporation gives beautiful crystals of shikimic acid; *Illicium anisatum* gives a very little quantity of small crystals and sometimes none at all. In case the fruit of *Illicium religiosum* were taken in fine powder, Collin (19) advises the microscopic examination of the vomitus or the content of the stomach for fragments of the epicarp and of the endocarp and for the external envelope of the seminal integument. The distinctive characteristics of these parts of the fruit are the striated cuticle lining the epicarp, the special configuration of the sclerenchyma cells of the endocarp, and the highly sinuous sclerenchyma cells of the external envelope of the seminal integument, which also contains crystals in its internal envelope.

Most of the properties of the two species of *Illicium* given above are of value only to the experts. It is self-evident that the laymen, who daily use the fruit of *Illicium anisatum* in the preparation of food and who, on account of their social condition, are less protected against all sorts of adulteration in spite of the Pure Food and Drug Law now in force, require the character which is decisive and is easily detected. Eykman noted that the fruit of *Illicium religiosum* may sometimes simulate the anislike odor and taste of *Illicium anisatum*, but its usual taste and odor of cubeb and laurel are in our opinion the most practical signs for laymen to use in the differentiation of one variety from the other. The detection of the odor and taste is facilitated by triturating or merely crushing one or two pods of the fruit.

Holmes (20) gives the differential properties of the fruits of the various species of *Illicium* as is shown in Table II.

TABLE II.—*Differential properties of the fruits of various species of Illicium.*

Species.	Carpels.	Taste.
Illicium anisatum	8	Of anise.
Illicium religiosum	8	Slightly that of laurel leaves.
Illicium parviflorum	8	Of sassafras.
Illicium floridanum	13	Of anise.
Illicium griffithii	13	Bitter and at the same time that of the leaves of laurel and of cubeb.
Illicium majus	13	Of mace.

It may be mentioned in passing that there are other species of *Illicium.* Mr. E. D. Merrill, botanist, Bureau of Science, has recently discovered in the Philippine Islands two indigenous species, *Illicium montanum* and *Illicium philippinense.*

THE ACTIVE PRINCIPLE AND PHARMACOLOGICAL ACTION OF ILLICIUM RELIGIOSUM

The chemical isolation of the poisonous principle of *Illicium religiosum* does not appear to have been the subject of many careful investigations. Eykman claimed to have succeeded in isolating from the fruit a colorless crystalline substance to which he ascribed the toxicity of *Illicium religiosum.* He provisionally named this substance "sikimine." When administered to a young dog in a dose of 12 milligrams, it produced the following symptoms:

After ten minutes: Disquiet, strong barking, twisting of the head and of the tongue in the opened mouth, scratching of the stomach and chest with the hinder extremities, afterward crossness. After fifteen minutes: Violent cramps of the muscles of the abdomen with disposition to vomit, tetanic convulsions with simultaneous extension of the four extremities, stronger flexion of the head toward the back, convulsive yawning, formation of foam, vomiting of a brown slimy mass, starting forward, sudden turning around of the entire body, afterward strong convulsions in the extremities, then paralysis, lastly collapse and death after three hours.

Langaard has carefully studied the actions of *Illicium religiosum* on rabbits, frogs, and fishes. It has been shown by him that all parts of the plant contained poison. From the results of his experiments with the alcohol-free alcoholic extract of the roots of the plant, he drew the following conclusions:

1. The poison produces convulsions by stimulating the medulla oblongata.

2. Before the appearance of the convulsions the reflex irritability in frogs is lowered, due to the stimulation of the inhibitory center in the brain. The spinal cord is not affected in the beginning, but the convulsions in the later stage of the poisoning arise from the heightened reflex excitability of the spinal cord.

3. The respiration is accelerated through the stimulation of the respiratory center.

4. Small quantities bring about slowing of the heart beat through the stimulation of the center and termination of the

vagus nerve. The irritability of the latter is depressed toward the end.

5. Small doses kill by paralyzing the respiration, while very large doses kill through paralysis of the heart.

We have not been successful in eliciting increased reflex irritability of the spinal cord in our experiments on monkeys, dogs, and cats in the interconvulsive period. There were convulsions and shaking of the entire body, but the animals did not respond to taps on the back, pinching of the tail, and moving the limbs—tests which promptly bring about convulsions in strychninized animals. The convulsions can be readily made to break out by causing the animals to struggle, for example, by lifting them up by the skin of the back. In fact it appeared that voluntary movements frequently precipitated the convulsions.

TREATMENT OF POISONING BY ILLICIUM RELIGIOSUM

The rational treatment of poisoning by *Illicium religiosum* will not be possible till the chemical and pharmacological reactions of its poisonous principle are definitely established. If the poison was introduced per orem, as is usually the case, the stomach should be rapidly evacuated, either by the use of apomorphine or of the stomach tube. Apomorphine offers decided advantages over the other medicinal emetics, on account of its ready action and the facility with which it can be administered, and lastly because it will produce emesis without causing further irritation of the stomach. After the stomach is evacuated, demulcent drinks or emollients may be administered. In our opinion liquid paraffin is the most preferable. It is not absorbable and is not changed by the digestive juices; hence its soothing effect will extend to the entire length of the alimentary tract. Eykman has used chloral hydrate to overcome the convulsions; Langaard considered it a life-saving remedy against the minimal lethal doses of *Illicium religiosum.* The efficacy of safe quantities of chloral hydrate against violent spinal convulsions is doubtful, since the action of such quantities on the spinal cord is little. We have used this drug in one of our cases, but from our experiments and the studies of Langaard, we believe that the best method of controlling the convulsions is by the careful inhalation of ether followed by the administration of chloral hydrate and sodium or potassium bromide; the bromides enhance the sedative effects of chloral hydrate. In the later stage of the poisoning, when paralytic symptoms predominate, these drugs are emphatically contraindicated. The treatment in this case is chiefly a struggle to keep up the working of the paralyzed respiratory center, the circulatory apparatus, and the organs of elimination.

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RINDERPEST IN SWINE WITH EXPERIMENTS UPON ITS TRANSMISSION FROM CATTLE AND CARABAOS TO SWINE AND VICE VERSA[1]

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TWO PLATES AND 10 TEXT FIGURES

INTRODUCTION

This investigation was commenced with the purpose of positively determining the rôle that pigs may play in the spread of rinderpest in the Philippine Islands.

During several years of experience in handling rinderpest in the field, Dr. Stanton Youngberg, chief veterinarian, Bureau of Agriculture, has frequently noticed, and has also received reports from veterinarians on rinderpest quarantine work in the field, that in localities where rinderpest is present pigs also develop an ailment practically simultaneous with the appearance of rinderpest in cattle and in carabaos.

There is one instance where it was practically proved that pigs were the cause of an outbreak of rinderpest among cattle, carabaos, and swine in the Philippines. In February, 1908, Doctor Youngberg was ordered to investigate an outbreak of disease on Romblon Island, which was causing serious losses among cattle and carabaos.

The disease proved to be rinderpest, and this was the first time it had made its appearance on that island. The outbreak was very virulent, and the mortality was high.

It was at first difficult to account for the introduction of the disease, as no cattle or carabaos had been imported there for a long time. However, it was ascertained that hogs had been and were being imported from Capiz, a province on Panay Island and situated some 120 kilometers south of Romblon. At that time rinderpest was prevalent in Capiz Province.

Information was obtained to the effect that hogs which had been kept in the same pens with those imported from Capiz sickened first and that many died; after this, other hogs in the village became infected, and then the carabaos and the cattle began to die. During this outbreak it was noticed that when a village was found infected with rinderpest hogs had previously died in considerable numbers.

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[1] Reprinted from *Phil. Agr. Rev.* (1916), 9, 288.

Although these facts were reported by Doctor Youngberg to the Bureau of Agriculture, the essential point of the possibility of hogs being the prime factor in the introduction of this disease was unfortunately overlooked by the authorities in charge.

The definite connection between rinderpest in cattle and in carabaos and the ailment in pigs was overlooked, because the mortality, as a rule, was not high among the pigs; the native pig is not a very thrifty-appearing individual; and swine plague, hog cholera, kidney-worm infestation, and *Balantidium coli suis* are very prevalent throughout the Islands. It was supposed that the pigs were suffering from one or more of the abovementioned diseases and that their unthriftiness was magnified by the fact that all animals in infected areas under quarantine were watched much more closely by the veterinarians and the livestock inspectors in charge than in rinderpest-free districts.

During 1914, while I was on vacation, hog cholera broke out among the pigs at the Bureau of Agriculture stock farm at Alabang, Rizal Province. Mr. Thomas L. Bean, assistant in the veterinary research laboratory, made the hog-cholera serum and immunized these pigs. Upon completing this work, Mr. Bean left six pigs hyperimmunized to hog cholera at the laboratory. These six pigs were exposed separately to cattle sick with rinderpest, as will be noted in experiments 43 to 48 in this paper, and each one developed symptoms similar to those caused by rinderpest in cattle.

There is but little literature upon the subject of rinderpest in swine, and this little is contradictory.

Carré and Fraimbault,(2) in the course of research work upon rinderpest in Indo-China, performed some experiments upon pigs. They demonstrated the possibility of transmitting rinderpest from pig to pig by contact, as well as by blood inoculation. They also demonstrated that it was possible to transmit rinderpest by blood inoculation from bull to pig and from pig to bull. Carré and Fraimbault also mention that M. Leblanc had found rinderpest among peccaries in 1886 and that Penning had announced the transmission of rinderpest to the wild boar.

Friedberger and Frohner(3) mention Pluning as having noticed rinderpest in swine in Sumatra and that the symptoms and lesions were the same as those found in cattle. The same authors mention Theiler as being unable to transmit rinderpest to swine by inoculating them with blood from rinderpest-sick cattle.

Hutyra and Marek(4) state that the question of rinderpest in swine is not definitely settled, undoubtedly basing their statement on the contradictory results obtained by different workers. They cite Carré and Fraimbault, Penning, and Theiler.

Jobling,(5) in his annual report for 1903, cites an experiment upon rinderpest in pigs in the Philippine Islands. In this experiment he injected a farm-bred hog with 5 cubic centimeters of virulent rinderpest blood from a bull. The hog developed typical symptoms of rinderpest. Blood was taken from it and injected into a hog and a bull. The hog that received this blood did not develop any symptoms of the disease. The bull that received the blood taken from the sick pig developed typical symptoms of rinderpest, but ran a mild course of the disease and was bled to death. Upon autopsy this animal presented lesions of rinderpest, but not so far advanced as is usually noticed.

In concluding this experiment Jobling says:

> This hog had the shortest incubation period of any animal I have seen, and I would have believed it to be suffering from some other disease had cow No. 46 not developed typical rinderpest after the regular incubation period.
>
> From this one series of experiments it would appear that while the hog may contract the disease and die, and its excreta may form a source of infection for cattle and carabaos, yet it is difficult for the sick hog to reinfect others of its kind. However, no definite conclusions could be based upon the evidence at my disposal, and the work will be continued when opportunity offers.

Jobling also mentions the possibility of wild hogs spreading the disease in the provinces.

It may be mentioned here, as will be noted in the following experiments, that hogs have as a rule a shorter incubation period and show a higher temperature during the course of the disease than do cattle and carabaos. The shortness of the incubation period may be accounted for by the facts that hogs are natural scavengers and when exposed to cattle and carabaos sick with rinderpest devour large quantities of fæces from these sick animals. In this way they immediately saturate their systems with large quantities of the virus, which is always present in abundance in the discharges of animals from the time of the initial rise in temperature to the time of recovery or death.

When virulent rinderpest blood taken from pigs was used in immunization work in the provinces, Doctor Youngberg noted that the virus was activated and better reactions were as a rule obtained than when the virulent blood was taken from cattle or carabaos. Also virulent blood from hogs mixed with virulent blood from cattle or carabaos had a tendency to activate the virus.

In a personal conversation Prof. Dr. T. Horiuchi, of the Bacteriological Institute of Formosa, informed me that he had seen pigs infected with rinderpest in that island. In his experience it was of rare occurrence that cattle became infected from pigs.

PIGS EXPOSED TO CATTLE SICK WITH RINDERPEST

The following ten experiments were designed to furnish information regarding the possibility of pigs contracting rinderpest through contact with cattle sick with the disease.

These exposures were conducted in two stalls, which will be designated as stall No. 1 and stall No. 2. These stalls were 10 feet long by 12 feet wide. Stall No. 1 had a cement floor, and stall No. 2 had a wooden floor. The sick cattle were tied in these stalls, and the pigs were unrestrained during the exposures.

All the animals used in these experiments were kept in quarantine for a certain length of time, which will be stated in each case. While in quarantine their temperatures were taken twice a day, morning and afternoon, and each day their general physical condition was noted.

The following abbreviations will be used in connection with the data on the experiments: D., diarrhœa; E. L., eating little; N. E., not eating; D., E. L., diarrhœa, eating little; D., N. E., diarrhœa, not eating; V. B., virulent blood.

EXPERIMENT 1

Pig 185.—Known history prior to the experiment: This animal was a native pig, 7 months old, purchased in Manila, and placed in quarantine at the laboratory March 2, 1915. On May 2, 1915, it was inoculated with 50 cubic centimeters of blood from a pig suffering from hog cholera. This blood had been passed through a Berkefeld N. filter. The animal developed a mild form of hog cholera and recovered.

May 21, 1915, pig 185 was exposed to bull 3906 in stall No. 1. This exposure was continued for three days.

History of bull 3906 during the exposure:
- May 21: Fifth day after the initial rise in temperature.
- May 21–23: D., N. E.
- May 24: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

May 25, pig 185 developed a rise in temperature, registering, in the afternoon, 40.1° C. This was four days after the initial exposure to bull 3906.

May 26, pig 185 was transferred to corral No. 1 to accomplish experiments 11 and 23.

May 27, this animal showed an afternoon temperature of 40.9° C., which was the highest temperature registered during the course of the disease. It was bled and the blood used in experiment 29.

May 28–31, N. E.

June 1–9, E. L.

This animal gradually recovered from rinderpest, but did not become thrifty, and was killed August 7, 1915.

EXPERIMENT 2

Pig. 207.—Known history prior to the experiment: This animal was a native pig, 7 months old, purchased in Manila and kept in quarantine twenty days before being used. At no time during this period did it have a high temperature or show any symptoms of sickness.

June 17, 1915, pig 207 was exposed to bull 3908, first day of temperature, and to bull 3926, sixth day of temperature. This exposure was made in stall No. 1 and was continued for five days.

History of bull 3908 during the exposure:
  June 17: First day of temperature, registering, a. m., 38.5° C., p. m., 39.3° C.
  June 22–25: D., N. E.
  July 4: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

History of bull 3926 during the exposure:
  June 17: Sixth day after the initial rise in temperature, N. E.
  June 18–20: D., N. E.
  June 21–22: D.
  This animal gradually recovered.

June 21, pig 207 developed a rise in temperature, registering, in the afternoon, 40.1° C.; this was four days after the initial exposure to bulls 3908 and 3926.

June 22, pig 207 was transferred to corral No. 1 to accomplish experiment 24.

June 24, this animal showed an afternoon temperature of 41.2° C., which was the highest temperature it developed during the course of the disease.

June 30 to July 2, this animal had a slight diarrhœa and ate but little.

The pig gradually recovered from rinderpest, but did not become thrifty, and was killed on August 7, 1915.

EXPERIMENT 3

Pig 208.—Known history prior to experiment: This animal was a native pig, 6 months old, purchased in Manila and kept in quarantine forty days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

July 19, 1915, pig 208 was exposed to bull 3939, fifth day of temperature, and to bull 3931, fourth day of temperature, in stall No. 1. This exposure was continued two days with bull 3939 and four days with bull 3931.

History of bull 3939 during the exposure:
  July 19: Fifth day of temperature, D., N. E.
  July 20: D., N. E.
  July 21: Died of rinderpest, presenting typical lesions of that disease upon autopsy.
History of bull 3931 during the exposure:
  July 19: Fourth day of temperature.
  July 21–22: N. E.
  July 22: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

July 23, pig 208 developed a rise in temperature, registering, in the afternoon, 40.4° C.; this was four days after the initial exposure to bulls 3939 and 3931.

July 27, pig 208 was removed to corral No. 1 to accomplish experiments 12 and 25. It presented a morning temperature of 39.9° C. and an afternoon temperature of 41.2° C.

July 31 to August 7, E. L.

August 3, it presented an afternoon temperature of 41.6° C., which was the highest temperature registered during the course of the disease.

August 8–11, D., E. L.

August 12, died of rinderpest, presenting typical lesions of that disease upon autopsy.

EXPERIMENT 4

Pig 215.—Known history prior to experiment: This animal was a native pig, 8 months old, purchased in Manila and kept in quarantine seventy-two days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

September 30, 1915, pig 215 was exposed to bull 3925, which was the third day after the bull's initial temperature was recorded. This exposure was continued for four days in stall No. 1.

History of bull 3925 during the exposure:
September 30: Third day of temperature.
October 1–3: D., N. E.
October 3: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

October 3, pig 215 developed a rise in temperature, registering, in the afternoon, 40.2° C.; this was three days after the initial exposure to bull 3925.

October 4, it presented an afternoon temperature of 40.9° C., which was the highest temperature registered during the course of the disease.

October 6–12, D., N. E.

October 13–16, D.

This animal gradually recovered from rinderpest, but did not become thrifty, and was killed December 4, 1915.

EXPERIMENT 5

Pig 213.—Known history prior to experiment: This animal was a native pig, 7 months old, purchased in Manila and kept in quarantine twenty-eight days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

August 17, 1915, pig 213 was exposed to bull 3928, first day of temperature, in stall No. 1.

History of bull 3928 during the exposure:
August 17: First day of temperature, p. m., 39.9° C.
August 18: Temperature, a. m., 39.6° C.; p. m., 40.8° C.
August 23: N. E.
August 24–29: D., N. E.
August 30–31: E. L.
This animal gradually recovered.

August 25, pig 213 presented a rise in temperature, registering, in the afternoon, 39.9° C.; this was eight days after the initial exposure to bull 3928.

This animal showed a rather high temperature until September 10, when its temperature subsided to normal and recovery was prompt. At no time during this period did the animal present any severe symptoms, such as diarrhœa or inappetence. The disease ran a mild course throughout, which is occasionally observed in cattle and carabaos.

EXPERIMENT 6

Pig 220.—Known history prior to experiment: This animal was a native pig, 7 months old, purchased in Manila and kept in quarantine forty-nine days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

October 9, 1915, pig 220 was exposed to bull 3938, second day of temperature, in stall No. 1. This exposure was continued for six days.

October 14, bull 3958, first day of temperature, was admitted to stall No. 1. This exposure was continued for eight days.

October 19, bull 3961, first day of temperature, was admitted to stall No. 1. This exposure was continued for three days.

History of bull 3938 during the exposure:

October 9: Second day of temperature, a. m., 39.9° C.

October 12–14: D., N. E.

October 15: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

History of bull 3958 during the exposure:

October 14: Initial rise in temperature, a. m., 39.3° C.; p. m., 40.4° C.

This animal ran a high temperature until October 22, when the temperature subsided to normal. It did not develop diarrhœa or inappetence, but ran a mild course of the disease.

History of bull 3961 during the exposure:

October 19: Initial rise in temperature, a. m., 39.6° C.; p. m., 40.6° C.

October 21: D.

October 22: D., N. E.

October 23: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

October 16, pig 220 showed a rise in temperature, registering, in the afternoon, 40.8° C.; this was seven days from the time of the initial exposure.

October 18 and 19, the afternoon temperatures were 40.8° and 40.9° C., respectively.

October 25, this animal's temperature had dropped to normal and recovery was prompt.

At no time did this animal present severe symptoms, such as diarrhœa and inappetence, but ran a mild course of the disease.

EXPERIMENT 7

Pig 240.—Known history prior to experiment: This animal was a native pig, 8 months old, purchased in Manila and kept in quarantine thirty-six days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

November 20, 1915, pig 240 was exposed for one day in stall No. 1 to bull 3972, which was on the sixth day after its initial rise in temperature.

Also to bull 3988 for three days in stall No. 1, this animal developing its initial rise in temperature on this day.

History of bull 3972 during the exposure:
  November 20: Sixth day of temperature, D., N. E.
  November 21: Died of rinderpest, presenting typical lesions of that disease upon autopsy.
History of bull 3988 during the exposure:
  November 20: Initial rise in temperature recorded; p. m. temperature, 41° C.
  November 25: This animal was bled to death to secure virulent blood for immunizing purposes.

November 23, pig 240 presented a rise in temperature, registering, in the afternoon, 40.2° C.; this was three days from the time of the initial exposure.

November 24–26, N. E.

November 26, transferred to stall No. 2 to be used in experiment 27.

November 27–28, D., N. E.

November 29, E. L.

This animal gradually recovered, but later contracted hog cholera.

EXPERIMENT 8

Pig 155.—Known history prior to exposure: This animal was a Berkshire sow about 10 months old; it was brought to the laboratory from the Bureau of Agriculture experiment station at Alabang on October 13, 1915, and was kept in quarantine fifty-two days before being used. During this period at no time did it have a high temperature or show any symptoms of sickness.

December 3, 1915, pig 155 was exposed for two days to bull 3981 in stall No. 2. At the beginning of the exposure this bull was on its sixth day of temperature.

Also to bull 3983 for three days in stall No. 2. At the beginning of exposure this bull was on the fifth day of temperature.

History of bull 3981 during the exposure:
  December 3: Sixth day of temperature, D., N. E.
  December 4: Died of rinderpest, presenting typical lesions of that disease upon autopsy.
History of bull 3983 during the exposure:
  December 3: Fifth day of temperature, D., N. E.
  December 4: D., N. E.
  December 5: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

December 12, pig 155 developed a morning temperature of 40.2° C.; this was nine days from the time of the initial exposure.

This animal had a high temperature until December 21, at which date the temperature subsided to normal.

This animal ran a mild course of the disease, never showing severe symptoms such as diarrhœa and inappetence.

EXPERIMENT 9

Pig 237.—Known history prior to exposure: This animal was a native pig, 5 months old, purchased in Manila and kept in quarantine seventy-five days before it was used. During this period at no time did it have a high temperature or show any symptoms of sickness.

December 29, 1915, pig 237 was exposed to bull 3966, which was on its fifth day of temperature. This exposure lasted two days in stall No. 2.

Also to bull 3976 for five days in stall No. 2, commencing on the initial rise of temperature in the bull.

History of bull 3966 during the exposure:
  December 29: Fifth day of temperature, D., N. E.
  December 30: Died of rinderpest, presenting typical lesions of that disease upon autopsy.
History of bull 3976 during the exposure:
  December 29: First day of temperature, a. m., 39° C.
  January 3, 1916: Bled to death for virulent blood to be used in immunization work.

January 2, 1916, pig 237 developed a morning temperature of 41.2° C.; this was four days after the initial exposure.

January 3, this pig was bled to death for virulent blood to be used in immunization work.

EXPERIMENT 10

Pig 242.—Known history prior to exposure: This animal was a native pig, 6 months old, purchased in Manila and kept in quarantine seventy-six days before it was used.

This animal arrived at the laboratory on October 15, 1915. On October 26 this pig had a high temperature, and on October 28 it developed a diarrhœa. The diarrhœa continued until November 9. The high temperature continued until November 16, when it subsided to, and remained, normal. This condition was diagnosed as hog cholera, as another pig, which had been kept in quarantine with this one, developed similar symptoms just prior to this time. It was killed, and at autopsy it presented good lesions of hog cholera.

January 30, 1916, pig 242 was exposed in stall No. 2 for three days to bull 3960, which was on its fourth day of temperature.

History of bull 3960 during the exposure:

January 30: Fourth day of temperature.
January 31: D., N. E.
February 1: Died of rinderpest, representing typical lesions of that disease.

February 2, 1916, pig 242 developed a morning temperature of 39.8° C., and an afternoon temperature of 41° C.; this was three days after the initial exposure.

February 4–5, E. L.

February 6–7, N. E.

February 7, died of rinderpest, presenting typical lesions of that disease upon autopsy.

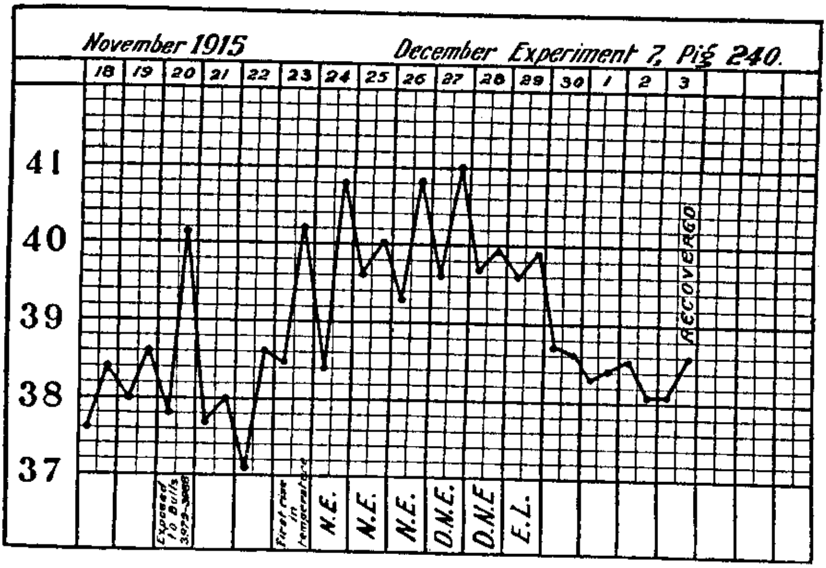

FIG. 1. Temperature chart of pig 240 exposed to bull.

In summing up the results of the ten preceding experiments on the exposure of pigs to cattle sick with rinderpest, it will be noted that all the pigs contracted a disease and developed symptoms characteristic of rinderpest in cattle and carabaos.

Three of the animals developed their initial rise in temperature on the third day, four on the fourth day, one on the seventh day, one on the eighth day, and one on the ninth day of exposure. From this it will be noted that the incubation period of rinderpest in pigs that have been exposed to cattle sick with that disease corresponds very closely to the incubation period in cattle and carabaos, in which it varies between three and ten days, averaging five days.

Of the ten pigs used, two, or 20 per cent, died of rinderpest. Four, or 40 per cent, recovered from the disease, but were unthrifty and were killed, although under normal conditions these animals would undoubtedly have been allowed to live. Of these four, one contracted hog cholera. Three, or 30 per cent, presented very mild symptoms, showing only a temperature reaction, and recovered promptly; these animals under normal field conditions would hardly have been suspected of being sick. One, or 10 per cent, was bled to death for virulent blood, leaving the final outcome as to the effect of the disease uncertain.

These 10 pigs were exposed to 19 head of cattle sick with rinderpest. Of these 19 sick cattle, 14, or approximately 73.7 per cent, died of rinderpest; three, or approximately 15.8 per cent, recovered; two, or approximately 10.5 per cent, were bled to death for virulent blood, leaving the final outcome as to the effects of the disease uncertain.

In comparing these figures, it will be noted that the mortality in cattle was 73.7 per cent against 20 per cent in pigs and that the recovery in cattle was 15.8 per cent against 70 per cent in pigs when the mild cases and those that recovered but were unthrifty are figured together.

Thus it will be seen that rinderpest in pigs, when contracted by exposure, presents a low mortality as compared with its effect upon cattle.

PIGS EXPOSED TO PIGS SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of pigs contracting rinderpest by contact with pigs sick with that disease.

These exposures were conducted in two different corrals which will be designated as corral No. 1 and corral No. 2. The dimensions of corral No. 1 were 14 by 30 feet, and of corral No. 2, 16 by 30 feet. Both corrals were built on the ground, and each contained a small lean-to shed to protect the animals from the weather.

The sick pigs and those placed in the corrals to be exposed were allowed the freedom of the place.

All the animals used in these experiments were kept in quarantine a certain length of time, which will be stated in each case. While in quarantine their temperatures were taken twice a day, morning and afternoon, and each day their general physical condition was noted.

EXPERIMENT 11

Pig 186.—Known history prior to the exposure: This animal was a native pig, 6 months old, purchased in Manila and brought to the laboratory and placed in quarantine on March 2, 1915.

On May 2, 1915, this animal was inoculated with 50 cubic centimeters of blood from a pig suffering from hog cholera. This blood had been passed through a Berkefeld N. filter. The animal developed a mild form of hog cholera and recovered. At no other time than when suffering from the mild attack of hog cholera did the animal have a rise in temperature or show any symptoms of sickness.

May 26, 1915, pig 186 was exposed to pig 185 in corral No. 1; this was the second day of temperature for pig 185. The exposure was continued for sixteen days.

History of pig 185 during the exposure:

Referring to experiment 1, it will be noted that pig 185 contracted the disease from bull 3906.

May 26: Temperature, a. m., 39.1° C.; p. m., 40.5° C.
May 27: Temperature, a. m., 40.2° C.; p. m., 40.9° C.
May 28–31: N. E.
June 1–9: E. L.

May 30, pig 186 developed a rise in temperature, registering, in the morning, 39.8° C.; in the afternoon, 40.6° C. This was four days after the initial exposure to pig 185.

May 31, afternoon temperature, 41.6° C., which was the highest temperature registered during the course of the disease.

June 1–4, D., N. E.

June 9–10, D., E. L.

June 15–16, D.

This animal gradually recovered, but did not become thrifty, and was killed August 7, 1915.

EXPERIMENT 12

Pig 212.—Known history prior to experiment: This animal was a native pig, 5 months old, purchased in Manila and kept in quarantine eight days before it was used. On the first day in quarantine it registered an afternoon temperature of 40.4° C., which was undoubtedly due to excitement, since the temperature subsided to normal on the following day and remained so until after it was used, and at no time prior to the exposure did it manifest any symptoms of sickness.

July 27, 1915, pig 212 was exposed to pig 208 in corral No.

1; this was the fifth day of temperature for pig 208. The exposure was continued for fifteen days.

History of pig 208 before and during the exposure:

Referring to experiment 3, it will be noted that pig 208 contracted the disease from bull 3939 and bull 3931.

July 27, fifth day of temperature, a. m., 39.9° C., p. m., 41.2° C.

July 31–August 7: E. L.

August 8–11: D., E. L.

August 12: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

August 1, pig 212 developed a rise in temperature, registering, in the morning, 40.6° C.; in the afternoon, 41.6° C. This was on the fifth day after the initial exposure to pig 208.

August 3–7, E. L.

August 9–11, D., N. E.

August 11, died of rinderpest, presenting typical lesions of that disease upon autopsy.

EXPERIMENT 13

Pig 218.—Known history prior to the experiment: This animal was a native pig, 8 months old, purchased in Manila and placed in quarantine at the laboratory on August 21, 1915, where it was kept forty-five days before it was used. At no time during this period did it have a high temperature or show any symptoms of disease.

October 5, 1915, pig 218 was exposed to pig 216 in corral No. 1; this was the third day of temperature for pig 216. The exposure was continued for one day.

History of pig 216 before and during the exposure:

Referring to experiment 17 it will be noted that pig 216 was inoculated with 6 cubic centimeters of virulent rinderpest blood from bull 3925.

October 5: D., N. E., and vomiting.

October 6: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

October 9, pig 218 developed a rise in temperature, registering, in the morning, 40.4° C.; in the afternoon, 41.6° C.; this was on the fourth day after the initial exposure to pig 216.

October 11–12, E. L.

October 13, died of rinderpest, presenting typical lesions of that disease upon autopsy.

In summing up the results of experiments 11, 12, and 13, on the exposure of susceptible pigs to pigs sick with a disease they had contracted from cattle sick with rinderpest, these pigs manifesting symptoms agreeing with the symptons of rinderpest in cattle and carabaos, and finally upon autopsy after death presenting lesions characteristic of that disease in cattle, it will be noted that the pigs exposed to these sick ones promptly developed a disease which possessed all the characteristics of rinderpest. Two of the animals developed a rise in temperature on the fourth day after the initial exposure and one on the fifth day, which time corresponds very closely with the incubation period of rinderpest in cattle and carabaos.

Of the three animals used, two, or $66\frac{2}{3}$ per cent, died, and one, or $33\frac{1}{3}$ per cent, recovered, which also agrees very closely with the effects of rinderpest on cattle and carabaos.

Although the animal in experiment 11 was finally destroyed on account of being unthrifty, under normal conditions in the Philippines, this animal undoubtedly would have been allowed to live.

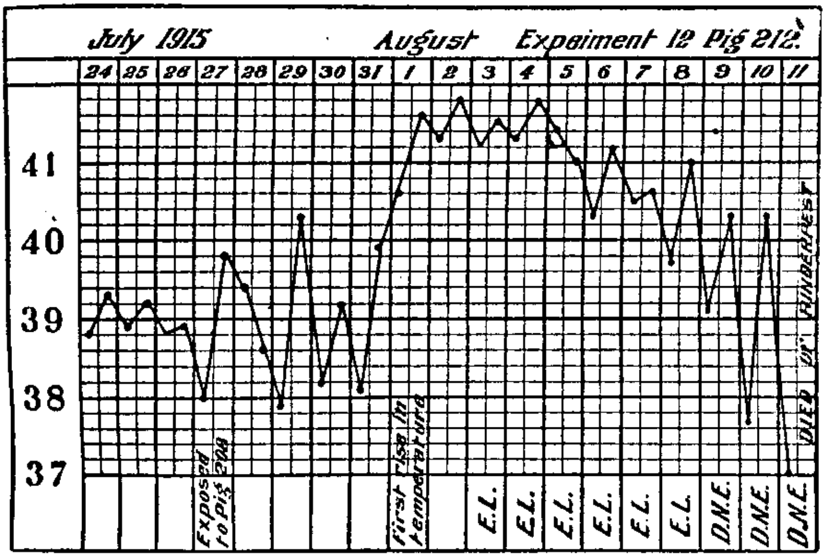

FIG. 2. Temperature chart of pig 212 exposed to pig.

In comparing the percentage of mortality in pigs exposed to pigs sick with rinderpest with that of pigs exposed to cattle sick with rinderpest, it appears that the virulence of the disease is increased for pigs by being passed through a pig.

RINDERPEST TRANSMITTED FROM PIG TO PIG BY MEANS OF THE CARETAKER

EXPERIMENT 14

Pig 214.—Known history prior to the experiment: This animal was a native pig, 8 months old, purchased in Manila and kept in quarantine thirty-one days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

August 21, 1915, pig 214 was placed in corral No. 1. This experiment was designed to determine the following: (1) Whether the virus of rinderpest was still alive in such a form as to give the disease to a susceptible pig when placed in the corral, since a pig ten days previously had died of rinderpest in this corral (this was in line with other experiments that have been performed at the laboratory on cattle and carabao,(6) in which it was proved that the virus of rinderpest was in such a condition that it was unable to cause the disease after a corral had been freed from sick animals for twenty-four hours); and (2) whether the virus of hog cholera was present in the corral, since a pig, which eleven days previously had died in this corral, showed simultaneous lesions of hog cholera and of rinderpest.

Before the completion of the experiment on pig 214 in this corral, added evidence was obtained on two other points: (1) The possibility of a pig contracting rinderpest after surviving hog cholera; and (2) the possibility of transmitting, by means of the caretaker, the virus of rinderpest from a pig sick with that disease to a susceptible pig.

History of corral No. 1 immediately previous to placing pig 214 therein:
- On August 11, which was eleven days previous to the beginning of this experiment, pig 212 (experiment 12) had died of rinderpest in this corral; this animal also showed lesions of hog cholera.
- On August 12, which was ten days previous to the beginning of this experiment, pig 208 (experiment 3) had died of rinderpest in this corral; this animal also showed lesions of hog cholera.

August 25, 1915, pig 214 showed a rise in temperature. Its afternoon temperature remained high, ranging between 40° and 40.8° C., until September 15, when it gradually subsided to normal. During this time, although the animal did not develop diarrhœa or show marked inappetence, it was unthrifty and had a mucopurulent discharge from its eyes, presenting symptoms of a mild attack of hog cholera.

October 5, 1915, pig 216 (experiment 26) was placed in corral No. 2. This corral was approximately 45 meters from corral No. 1, in which pig 214 was kept at that time. Pig 216 was on its second day of temperature, D., N. E. on this date, and it died of rinderpest on October 6.

Pig 218 (experiment 13) developed its first rise of temperature on October 9 and died of rinderpest four days later. These two animals were cared for by the same man that took care of pig 214 in corral No. 1.

October 14, 1915, pig 214 developed an afternoon temperature of 40.5° C. This was nine days after pig 216 was placed in corral No. 2 and eight days after its death, and five days after the initial rise in temperature of pig 218, which was also kept in corral No. 2.

October 16–18, N. E.

October 18, died of rinderpest, showing typical lesions of that disease upon autopsy.

In summing up the results of experiment 14, it will be noted that pig 214 did not contract rinderpest in corral No. 1 which had contained a pig that died of rinderpest ten days previous to the admission of pig 214 to the corral. It will also be noted that pig 214 did have a temperature and showed mild symptoms of hog cholera, which it possibly contracted in corral No. 1. It will be noted also that pig 214 did finally contract rinderpest and died of that disease and that the disease was undoubtedly brought to it by means of the caretaker.

Also it will be noted that a pig can suffer from an apparently mild attack of hog cholera and recover from that disease and later contract rinderpest and die.

PIGS INOCULATED WITH VIRULENT BLOOD FROM PIGS SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of pigs contracting rinderpest by inoculating them with blood taken from pigs sick with rinderpest. The animals that were inoculated were kept in screened stalls in a shed, which was free from disease, until they developed a rise in temperature, at which time they were removed.

EXPERIMENT 15

Pig 206.—Known history prior to the experiment: This animal was a native pig, 6 months old, purchased in Manila on May 28, 1915, and placed in quarantine.

May 29, 1915, pig 206 was inoculated with 10 cubic centimeters of virulent blood from pig 185.

History of pig 185 at the time the virulent blood was drawn from it:

May 29: Fourth day of temperature, N. E. The a. m. temperature was 39.4° C.; p. m. temperature, 40.1° C. For complete history see experiment 1.

June 2, pig 206 developed a rise in temperature, registering, in the afternoon, 40.2° C.; this was four days after inoculation.

June 3, this animal died. Autopsy did not reveal any marked lesions.

EXPERIMENT 16

Pig 219.—Known history prior to the experiment: This animal was a native pig, 9 months old, purchased in Manila and kept in quarantine forty-five days before it was used, and at no time during this period did it have a high temperature or show any symptoms of sickness.

October 5, 1915, pig 219 was inoculated with 2 cubic centimeters of virulent blood from pig 215.

History of pig 215 at the time the virulent blood was drawn from it:
October 5: Second day after the initial rise in temperature, a. m. temperature, 39.1° C.; p. m. temperature, 40.7° C. For complete history see experiment 4.

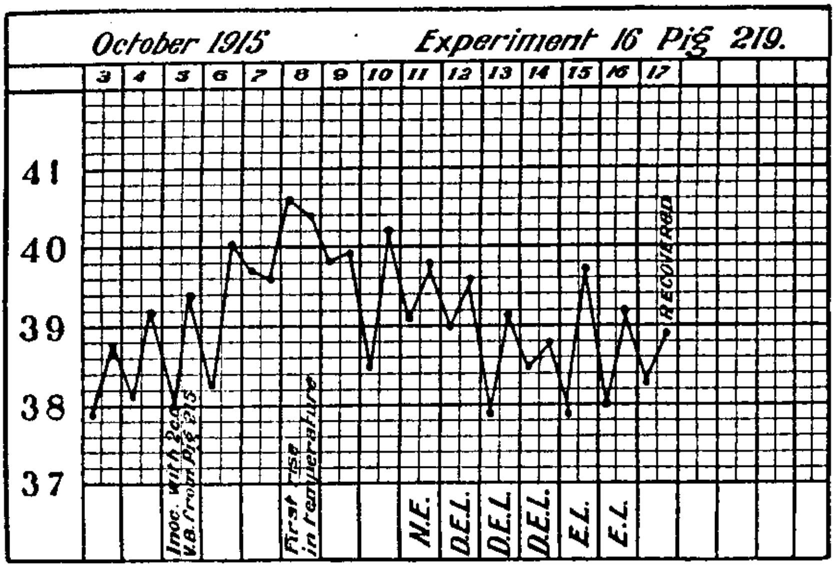

FIG. 3. Temperature chart of pig 219 inoculated with blood from pig.

October 8, pig 219 presented a rise in temperature registering, in the morning, 40.6° C.; in the afternoon, 40.4° C.; this was three days after the inoculation.

October 11, N. E.

October 12–14, D., N. E.

October 15–16, E. L.

This animal gradually recovered, but did not become thrifty and was killed December 4, 1915.

In summing up the results of experiments 15 and 16, it can not be stated with certainty that pig 206 used in experiment 15 contracted rinderpest, on account of its early death and the absence of the lesions that are to be expected in the early stage of the disease and which have been found in cattle and carabaos under similar conditions. However, the animal had an incubation period that corresponded to that of rinderpest.

In experiment 16, pig 219 ran a typical course of rinderpest and recovered with the after effects of being unthrifty, which condition is frequently noticed in pigs after they have recovered from rinderpest.

PIGS INOCULATED WITH VIRULENT BLOOD FROM CATTLE SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of pigs contracting rinderpest by inoculating them with blood taken from cattle sick with rinderpest. The animals that were inoculated were kept in screened stalls in a shed, which was free from disease, until they developed a rise in temperature, at which time they were removed.

EXPERIMENT 17

Pig 216.—Known history prior to the experiment: This animal was a native pig, 7 months old, purchased in Manila and kept in quarantine seventy-two days before it was used. At no time during this period did it develop a temperature or show any symptoms of disease.

September 30, 1915, pig 216 was inoculated with 6 cubic centimeters of virulent blood from bull 3925, which was on its second day of temperature.

History of bull 3925:
- September 25: Inoculated with 200 cubic centimeters of virulent blood from bull 3937.
- September 28: First rise in temperature, p. m., 40.4° C.
- September 30: Second day of temperature, blood was taken to inoculate pig 216.
- October 1–3: D., N. E.
- October 3: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

October 3, pig 216 developed a rise in temperature, registering, in the morning, 40.5° C.; in the afternoon, 41.1° C.; this was three days after the inoculation.

October 5, D., N. E., and vomiting.

October 6, died of rinderpest, presenting marked lesions of that disease upon autopsy. This pig was used in exposure experiments 13 and 26.

EXPERIMENT 18

Pig 241.—Known history prior to the experiment: This animal was a native pig 6 months old, purchased in Manila and kept in quarantine one hundred fifteen days before it was used. It developed a high temperature on October 26, 1915, and was taken out of the quarantine shed and isolated. It developed a diarrhœa October 28, which lasted until November 7, when the diarrhœa subsided, and the temperature gradually returned to normal. During this period the animal presented a clinical picture of a mild case of hog cholera. November 26 pig 241 was returned to the quarantine shed, where it remained until it was used, and did not show a rise of temperature or any symptoms of sickness.

February 7, 1916, pig 241 was inoculated with 25 cubic centimeters of virulent rinderpest blood drawn from bull 3962, which was on its third day of temperature. Pig 241 was placed in a screened stall in a shed free from rinderpest.

History of bull 3962:
- February 1, 1916: Bull 3962 was inoculated with 100 cubic centimeters of virulent rinderpest blood from bull 3960.
- February 4: Bull 3962 developed a rise in temperature, registering, a. m., 39.8° C.; p. m., 40.6° C.
- February 5–8: D., N. E.
- February 8: This animal presented a subnormal a. m. temperature of 35.8° C., and died, presenting typical lesions of rinderpest upon autopsy.
- On the day the blood was drawn to inoculate into pig 241, this bull had diarrhœa and was not eating.

February 10, pig 241 developed a rise in temperature, registering, in the morning, 40° C.; in the afternoon, 41.3° C.; this was the third day after inoculation.

February 13, N. E.

Pig 241 died during the night of February 13 and presented typical lesions of rinderpest upon autopsy.

EXPERIMENT 19

Pig 239.—Known history prior to the experiment: This animal was a native pig, 8 months old, purchased in Manila and kept under observation one hundred twenty-two days before it was used. It developed a high temperature October 27, 1915, and was taken out of the quarantine shed and isolated. It developed a diarrhœa October 28, which continued until November 7, at which time the diarrhœa subsided, and the temperature returned to normal. During this period the animal presented a clinical picture of suffering from a mild attack of hog cholera. November 26 pig 239 was returned to the quarantine shed, where it remained until it was used, and did not develop a rise in temperature or show any symptoms of sickness during this period.

February 14, 1916, pig 239 was inoculated with 20 cubic centimeters of blood from bull 3993, which had been bled to death February 13 for virulent blood to be used in immunization work. The blood that was inoculated into the pig had been kept twenty-four hours in the ice box.

History of bull 3993 from which the blood was taken:

February 5, 1916: Drenched with 100 cubic centimeters of urine from pig 242 (experiment 35).
February 9: First rise of temperature, registering, p. m., 40.5° C.
February 12–13: E. L.
February 13: Bled to death for virulent blood to be used in immunization work.

Pig 239 did not develop a rise in temperature or show any symptoms of sickness from this injection and afterward was exposed in shed No. 3, where the animals sick with rinderpest were kept, and continued to give the same negative results.

EXPERIMENT 20

Pig 154.—Known history prior to the experiment: This animal was a Berkshire, about 1 year old; it was brought to the laboratory from the Bureau of Agriculture experiment station at Alabang on October 13, 1915, and was kept in quarantine at the laboratory one hundred thirty-two days before it was used. During this period the animal did not show a rise in temperature. January 10–11, 1916, it had diarrhœa, which was undoubtedly due to dietetic conditions.

February 23, 1916, pig 154 was inoculated with 8 cubic centimeters of blood from cow 3995, which had been bled to death on February 21 for virulent blood to be used in immunization work. The blood that was inoculated into pig 154 had been kept forty-eight hours in the ice box.

History of cow 3995 from which the blood was taken:

February 11, 1916: This animal was exposed to pig 241 (experiment 28).
February 17: First rise in temperature, registering, p. m., 40.2° C.
February 20: D., E. L.
February 21: D., N. E. On this date it was bled to death for virulent blood to be used in immunization work.

Pig 154 did not develop a rise in temperature or show any symptoms of sickness from this injection. It was later exposed in shed No. 3, where animals sick with rinderpest were kept, and it continued to give the same negative results.

In summing up the results of experiments 17, 18, 19, and 20, it will be noted that the pigs used in experiments 17 and 18 had an incubation period of three days and ran a severe course of the disease and died, presenting typical lesions of rinderpest upon autopsy.

Both of the pigs used in experiments 19 and 20 ran a high temperature and developed diarrhœa previous to being used in these experiments. At the time they were sick, it was thought that they were suffering from hog cholera; however, it is possible that they were suffering from a mild form of rinderpest, as it is almost impossible to distinguish the two diseases in pigs, unless autopsy is made or their blood is inoculated into susceptible cattle. These animals did not contract rinderpest when inoculated with virulent blood from cattle, and they did not contract rinderpest by subsequent exposures. The blood with which they were injected was proved to be virulent, as in both cases it was used in immunizing work with good results.

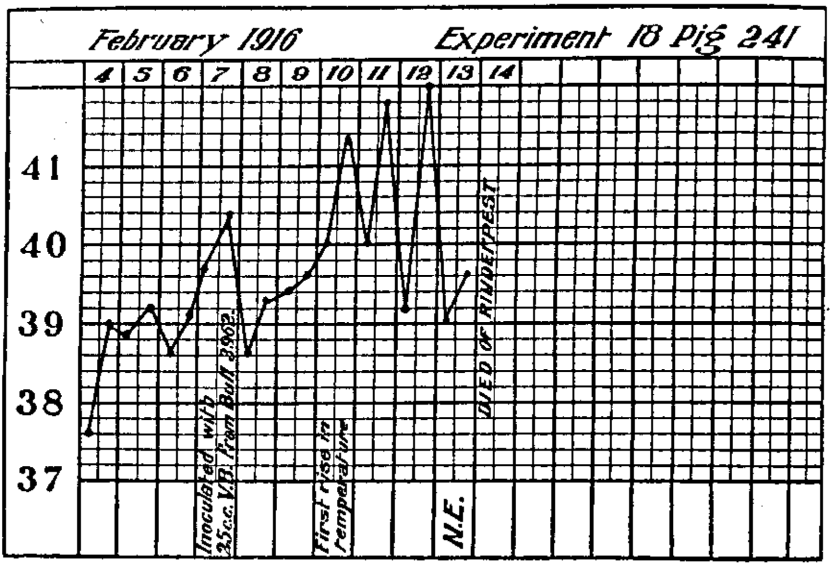

FIG. 4. Temperature chart of pig 241 inoculated with blood from bull.

Hence pigs 239 and 154 were either naturally immune to rinderpest or they had suffered from a previous attack of rinderpest and had acquired immunity.

PIGS INOCULATED WITH VIRULENT BLOOD FROM CATTLE AND PIGS SICK WITH RINDERPEST

The following experiment was designed to furnish information regarding the possibility of pigs contracting rinderpest when inoculated with a mixture of virulent rinderpest blood from cattle and pigs.

EXPERIMENT 21

Pig 244.—Known history prior to the experiment: This animal was a native pig, 6 months old, purchased in Manila and kept in quarantine seventy-eight days before it was used. At no time during this period did it have a high temperature or show any symptoms of sickness.

January 4, 1916, pig 244 was inoculated with a mixture of 25 cubic centimeters of virulent rinderpest blood from bull 3976 and pig 237.

History of bull 3976 from which blood was taken:

December 28, 1915: Bull 3976 was inoculated with 100 cubic centimeters of virulent rinderpest blood procured from a sick carabao in Pampanga Province.

December 31: First rise in temperature, registering, a. m., 39.1° C.

January 3, 1916: Bled to death for virulent blood to be used in immunizing work.

For history of pig 237, from which blood was taken, see experiment 9.

January 7, pig 244 developed a rise in temperature, registering, in the morning, 39.8° C.; in the afternoon, 40.6° C.; this was three days after the inoculation.

January 10, this animal's afternoon temperature was 41.3° C., which was the highest temperature registered during the course of the disease.

January 10–24, D., E. L.

January 24, died of rinderpest, presenting good lesions of that disease upon autopsy.

In summing up the results of this experiment it will be noted that a pig will contract rinderpest when inoculated with a mixture of virulent rinderpest blood from cattle and pigs.

PIGS DRENCHED WITH VIRULENT BLOOD FROM CARABAOS SICK WITH RINDERPEST

The following experiment was designed to furnish information regarding the possibility of pigs contracting rinderpest when given virulent rinderpest blood from carabaos by drench.

EXPERIMENT 22

Pig 243.—Known history prior to the experiment: This animal was a native pig, 6 months old, purchased in Manila and kept in quarantine ninety-two days before it was used. At no time during this period did it register a high temperature. On January 10 and 11 it had diarrhœa, which immediately subsided and was undoubtedly due to dietetic conditions.

January 18, 1916, pig 243 was drenched with 50 cubic centimeters of virulent rinderpest blood, procured from a carabao sick with rinderpest from Pampanga Province.

January 22, pig 243 presented a rise in temperature, registering, in the afternoon, 40.2° C.; this was four days after it had received the drench.

January 23, morning temperature, 41.0° C.

January 24, bled to death for virulent blood to be used in immunization work.

In summing up the results of this experiment, it will be noted that a pig will contract rinderpest when drenched with virulent rinderpest blood taken from a carabao sick with rinderpest.

CATTLE EXPOSED TO PIGS SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of cattle contracting rinderpest when exposed to pigs sick with rinderpest.

The experiments were conducted in corrals Nos. 1 and 2 and in stall No. 1.

The cattle used in these experiments were obtained from Batan Island. As, to our knowledge, there has never been any rinderpest on this island, these cattle are highly susceptible.

EXPERIMENT 23

Bull 3908.—Known history prior to the experiment: Native Batanes bull, 5 years old, received at the laboratory and placed in quarantine March 13, 1915. This animal was kept under observation seventy-four days before it was used, and at no time during this period did it present a high temperature or develop any symptoms of sickness.

May 26, 1915, bull 3908 was placed in corral No. 1 with pig 185, which was on its second day of temperature (see experiment 1), and the exposure was continued for fifteen days.

May 30, bull 3908 was also in contact with pig 186, first day of temperature (see experiment 11), and the exposure was continued for twelve days.

(For history of pigs 185 and 186 see experiments 1 and 11.)

Bull 3908 did not develop any symptoms from these exposures.

June 10, bull 3908 was inoculated with 200 cubic centimeters of virulent rinderpest blood from bull 3840 (see experiment 29).

June 17, first rise in temperature, registering, in the afternoon, 39.3° C., which was the highest temperature during the course of the disease; this was seven days after the inoculation.

June 15–20, D. It will be noted that the animal had a diarrhœa three days before its slight rise in temperature.

June 21–25, D., N. E.

June 26–July 3, D., E. L.

July 4, died of rinderpest, presenting good lesions of that disease upon autopsy.

EXPERIMENT 24

Bull 3924.—Known history prior to the experiment: Native Batanes bull, 3 years old, received at the laboratory and placed in quarantine April 13, 1915. This animal was kept under observation seventy days before it was used, and at no time during this period did it present a high temperature or show any symptoms of sickness.

June 22, 1915, bull 3924 was exposed in corral No. 1 to pig 207, which was on the second day of its temperature (see experiment 2). This exposure was continued twenty-two days.

History of pig 207 (see experiment 2):
- June 22: Temperature, p. m., 40.8° C.
- June 23: Temperature, p. m., 40.9° C.
- June 24: Temperature, p. m., 41.2° C.
- June 25: Temperature, p. m., 40.8° C.
- June 30–July 2: D.
- This animal gradually recovered, but never became thrifty.

Bull 3924 did not show a rise in temperature or present any symptoms of sickness during this exposure.

July 3, bull 3924 was inoculated with 100 cubic centimeters of virulent rinderpest blood taken from a carabao in Imus.

July 8, inoculated with 10 cubic centimeters of virulent rinderpest blood from bull 3930.

July 11, bull 3924 presented an afternoon temperature of 40° C. This was the highest and only rise in temperature this animal showed.

July 17, inoculated with 5 cubic centimeters of blood taken from bull 3929, which showed *Anaplasma* in its blood.

July 21–22, D.

September 13–14, D.

September 17, died, the cause of death not being ascertained.

EXPERIMENT 25

Bull 3935.—Known history prior to the experiment: Native Batanes bull, 2 years old, received at the laboratory and placed in quarantine June 16, 1915. This animal was kept under observation forty-one days before it was used, and at no time during this period did it present a high temperature or show any symptoms of sickness.

July 27, 1915, bull 3935 was exposed in corral No. 1 to pig 208, which was on its fifth day of temperature. This exposure was continued for sixteen days. This bull was also exposed on August 1 to pig 212 on the first day of temperature. This exposure was continued for eleven days.

History of pig 208 (see experiment 3):
- July 27: It presented an a. m. temperature of 39.5° C. and a p. m. temperature of 41.2° C.
- July 31–August 7: E. L.
- August 3: It presented a p. m. temperature of 41.6° C., which was the highest temperature registered during the course of the disease.
- August 8–11: D., E. L.
- August 12: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

History of pig 212 (see experiment 12):
- August 1: Temperature, a. m., 40.6° C.; p. m., 41.6° C.
- August 3–7: E. L.
- August 9–11: D., N. E.
- August 11: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

Bull 3935 did not show a rise in temperature or develop any symptoms of sickness from these two exposures.

September 16, 1915, bull 3935 was inoculated with 100 cubic centimeters of virulent rinderpest blood procured from a sick carabao in Pampanga Province.

September 20, bull 3935 presented a morning temperature of 39.6° C. and was bled to death for virulent blood to be used in immunization work.

EXPERIMENT 26

Bull 3886.—Known history prior to the experiment: Native Batanes bull, 3 years old, received at the laboratory and placed in quarantine November 7, 1914. This animal was kept under observation three hundred thirty-two days before it was used in this experiment, and at no time during this period did it have a high temperature or show any symptoms of sickness.

It had been used in two previous experiments which were as follows:

February 19, 1915, inoculated with 10 cubic centimeters of virulent rinderpest blood, which had been heated at 58° C. in a water bath for one hour. The animal did not develop any reaction from this.

August 28, 1915, this animal was inoculated with a culture made from rinderpest blood. The animal did not develop any reaction from this.

October 5, 1915, bull 3886 was exposed in corral No. 2 to pig 216, which was on the second day of temperature. This exposure lasted one day. This bull was also exposed on October 9 to pig 218, which was on the first day of temperature. This exposure was continued for nine days.

This bull was also exposed by means of the caretaker on October 13 to pig 214. This exposure was continued for five days.

History of pig 216 (see experiment 17):
- October 5: D., N. E., and vomiting.
- October 6: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

History of pig 218 (see experiment 13):
- October 9: First rise in temperature, registering, a. m., 40.4° C.; p. m., 41.6° C.
- October 11–12: E. L.
- October 13: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

History of pig 214 (see experiment 14):
- October 14: First rise in temperature.
- October 16–18: N. E.
- October 18: Died of rinderpest, showing typical lesions of that disease upon autopsy.

October 20, bull 3886 developed a rise in temperature, registering, in the afternoon, 40.2° C.

October 25–26, N. E.

October 27–30, D., N. E.

October 31–November 7, D., E. L.

November 7–11, D., N. E.

November 12, died of rinderpest, presenting typical lesions of that disease upon autopsy.

EXPERIMENT 27

Bull 3979.—Known history prior to the experiment: Native Batanes bull, 1 year and 4 months old, received at the laboratory and placed in quarantine October 23, 1915. This animal was kept under observation thirty-four days before it was used, and at no time during this period did it have a high temperature or show any symptoms of sickness.

November 26, 1915, bull 3979 was exposed four days in stall No. 2 to pig 240, which was on its third day of temperature.

History of pig 240 (see experiment 7):
November 26: N. E. Temperature, p. m. 40.8° C.
November 27–28: D., N. E.
November 29: E. L.
This animal gradually recovered, but later contracted hog cholera.

Bull 3979 presented no reaction to this exposure.

December 9, 1915, bull 3979 was inoculated with 150 cubic centimeters of virulent blood from bull 3963.

December 12, first rise in temperature.

December 13–14, N. E.

December 14, bled to death for virulent blood to be used in immunization work.

EXPERIMENT 28

Cow 3995.—Known history prior to the experiment: Native Batanes cow, 13 years old, received at the laboratory and placed in quarantine January 19, 1916. This animal was kept under observation twenty-three days before it was used, and at no time during this period did it have a high temperature or show any symptoms of sickness.

February 11, 1916, cow 3995 was exposed for four days in stall No. 2 to pig 241, which at the time of the exposure was on its second day of temperature.

History of pig 241 (see experiment 18):
February 11: Temperature, a. m., 40° C.; p. m., 41.8° C.
February 12: Temperature, a. m., 39.2° C.; p. m., 42° C.
February 13: N. E.
Pig 241 died during the night of February 13 and presented typical lesions of rinderpest upon autopsy.

February 17, cow 3995 presented a rise in temperature, registering, in the afternoon, 40.2° C.

February 18, temperature, morning, 39.2° C.; afternoon, 40.7° C.

February 20, D., E. L.

February 21, D., N. E., bled to death for virulent blood to be used in immunization work.

In summing up the results obtained in experiments 23 to 28, inclusive, it will be noted that cattle can contract rinderpest from pigs by being exposed by contact in a corral or stall to pigs sick with rinderpest, although the transmission of the disease does not appear to be so constant as by other methods; the reason for this has not been ascertained.

In experiment 23 it will be noted that bull 3908 and pig 186 (experiment 11) were exposed in the same corral and at the same time to pig 185 and that pig 186 contracted the disease but the bull was unaffected. It will also be noted that bull 3840 (experiment 29) was inoculated with blood from pig 185 on May 27 and contracted rinderpest from this blood, which proves that the blood of pig 185 was virulent when bull 3908 was exposed to it. It will also be noted that bull 3908 was exposed to pig 186 through the entire course of the disease, including the incubation period, and remained unaffected. Finally it will be noted that bull 3908 was inoculated with virulent rinderpest blood which was taken from bull 3840 and that bull 3908 contracted rinderpest from this inoculation and died. Thus, by inoculation, bull 3908 was killed with the same virus to which it had been exposed in pig 185 without effect.

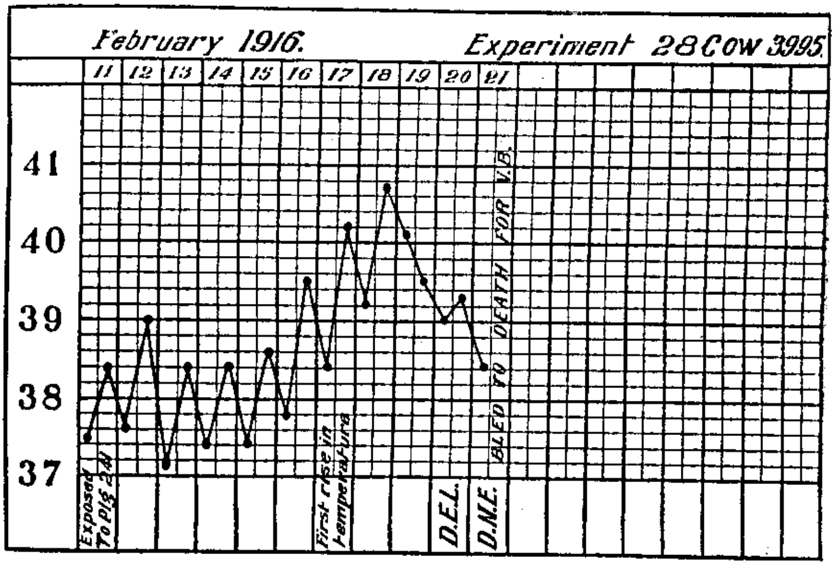

FIG. 5. Temperature chart of cow 3995 exposed to pig.

In experiment 24 bull 3924 was either naturally immune to rinderpest, a condition of rare occurrence, or it contracted a very mild type of disease from the exposure to pig 207 and thereby developed an active immunity, since it was unaffected by subsequent inoculations of virulent rinderpest blood.

In experiment 25 it will be noted that bull 3935 and pig 212 (experiment 12) were exposed in the same corral and at the same time to pig 208 and that pig 212 contracted the disease but bull 3935 remained unaffected. It will also be noted that bull 3935 was exposed to pig 212 through the entire course of the disease, including the incubation period, and that the bull remained unaffected. However, when this animal was inoculated with virulent blood, it contracted rinderpest, which proves that it was susceptible during the exposures, but for some unknown reason it failed to contract rinderpest by contact with the pigs.

In experiment 26 bull 3886 contracted rinderpest and died of that disease by being exposed to pigs sick with rinderpest, which proves that it is possible for cattle to contract rinderpest by being exposed to pigs sick with that disease.

Experiment 28 gives proof similar to that of experiment 26, showing that it is possible for cattle to contract rinderpest from pigs by exposure.

CATTLE INOCULATED WITH BLOOD FROM PIGS SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of cattle contracting rinderpest when inoculated with blood from pigs which were sick with that disease.

The cattle used in these experiments were of a similar type to those used in the preceding experiments.

EXPERIMENT 29

Bull 3840.—Known history prior to the experiment: Native Batanes bull, 4½ years old, received at the laboratory and placed in quarantine September 28, 1914. This animal was kept under observation for two hundred forty-one days before it was used in this experiment.

On October 6, 1914, this animal was inoculated with 50 cubic centimeters of blood taken from a sick animal at Calamba. Bull 3840 did not present any reaction to this inoculation.

October 18, 19, and 20 this animal presented afternoon temperatures of 39.9°, 40°, and 39.9° C., respectively. At no other time during this period of observation did it present a high temperature, and at no time did it show any symptoms of sickness.

May 27, 1915, bull 3840 was inoculated with 20 cubic centimeters of virulent blood from pig 185. (For history of pig 185 see experiment 1.)

May 30, bull 3840 presented a rise in temperature, registering 40° C. in the afternoon.

June 5–6, N. E.

June 7–9, D., E. L.

This animal gradually recovered, and was later used in making hyperimmune rinderpest serum.

August 19, bull 3840 received subcutaneously 1,000 cubic centimeters of virulent rinderpest blood from bull 3928. This injection caused a slight rise in temperature on the following day, but the animal suffered no other disturbance.

September 20, bull 3840 received subcutaneously 1,000 cubic centimeters of virulent rinderpest blood from bull 3935. This injection caused practically no reaction.

EXPERIMENT 30

Bull 3937.—Known history prior to the experiment: Native Batanes bull, 4 years and 9 months old, received at the laboratory and placed in quarantine June 16, 1915. This animal was kept under observation forty-eight days before it was used, and at no time during this period did it have a high temperature or show any symptoms of sickness.

August 3, 1915, bull 3937 was inoculated with 0.5 cubic centimeter of blood from pig 212.

History of pig 212 (see experiment 12):
August 3: E. L.

Bull 3937 presented no reaction to this inoculation.

September 20, bull 3937 was inoculated with 100 cubic centimeters of virulent rinderpest blood from bull 3935.

September 23, rise in temperature.

September 26–27, D., N. E.

September 27, died of rinderpest, presenting typical lesions of that disease upon autopsy.

EXPERIMENT 31

Bull 3839.—Known history prior to the experiment: Native Batanes bull, 4 years old, received at the laboratory and placed in quarantine September 28, 1914. This animal was kept under observation three hundred seventy-two days before it was used in this experiment.

October 10, 1914, this animal presented an afternoon temperature of 40° C. and was isolated from the quarantine shed.

October 11, D.

October 12, D., E. L.

October 18, this animal was returned to the quarantine shed, as its temperature and physical appearance were normal. The temperature and diarrhœa were undoubtedly due to dietary conditions.

May 4, 1915, this animal developed a diarrhœa which lasted but one day.

May 21, 22, and 23, this animal again developed a diarrhœa, but did not present a high temperature and soon returned to normal.

August 24, 1915, bull 3839 was inoculated with a culture in an experiment on the cultivation of the rinderpest virus, but the animal failed to show any reaction to this inoculation.

October 5, 1915, bull 3839 was inoculated with 3 cubic centimeters of virulent rinderpest blood from pig 216.

History of pig 216 (see experiment 17):

October 5: D., N. E., vomiting.

October 6: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

October 9, bull 3839 presented a morning temperature of 39.8° C.

October 12, 13, and 14, D., N. E.

October 15, died of rinderpest, presenting typical lesions of that disease upon autopsy.

EXPERIMENT 32

Cow 3958.—Known history prior to the experiment: Native Batanes cow, 2 years old, received at the laboratory and placed in quarantine October 1, 1915. This animal was kept under observation ten days before it was used.

October 5, D. The animal did not possess a temperature, however, and the bowels were normal the following day.

October 10, 1915, cow 3958 was inoculated with 50 cubic centimeters of virulent rinderpest blood from pig 217.

History of pig 217:

Contracted disease by exposure to sick pigs.

October 8: First rise in temperature, registering, a. m., 39.7° C.; p. m., 41° C.

October 11: D., E. L.

October 14, cow 3958 presented a morning temperature of 39.3° C.; afternoon temperature, 40.4° C.

This animal showed a high temperature until October 22, when its temperature subsided to normal. It did not develop inappetence or diarrhœa during the course of the disease and was proved immune by subsequent exposures in the shed where the animals sick with rinderpest were kept.

In summing up the results obtained in experiments 29 to 32, inclusive, it will be noted that cattle readily contract rinderpest when inoculated with blood taken from pigs sick with that disease.

In experiment 30 the bull failed to contract the disease when inoculated with 0.5 cubic centimeter of blood. Although this is a very small amount, yet such a quantity is usually sufficient to give the disease, and a much smaller quantity has been demonstrated to transfer the disease when the virulent blood was taken from a sick bull. (1)

In experiment 32 cow 3958 was inoculated with blood from pig 217 to ascertain whether the pig had contracted rinderpest or hog cholera in the quarantine shed, the result showing that the pig had contracted rinderpest.

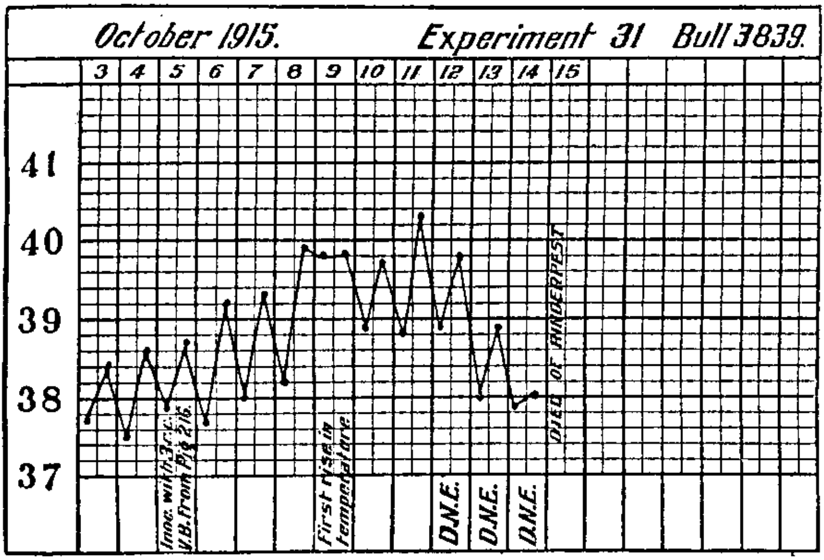

FIG. 6. Temperature chart of bull 3839 inoculated with blood from pig.

CATTLE INOCULATED WITH A MIXTURE OF BLOOD FROM PIGS AND CATTLE SICK WITH RINDERPEST

The following experiment was designed to furnish information regarding the possibility of cattle contracting rinderpest when inoculated with a mixture of blood from pigs and cattle sick with rinderpest, as it was desired in several instances to obtain more blood for immunization work than would be furnished by one bull. Thus, if the virus maintained its potency by mixing pig and cattle blood, pigs frequently could be bled to death to supply the demand.

EXPERIMENT 33

Cow 3987.—Known history prior to the experiment: Native Batanes cow, 1 year and 2 months old, received at the laboratory and placed in quarantine October 23, 1915. This animal was kept under observation forty-three days before it was used in this experiment.

December 31, 1915, D.

January 1, 1916, D.

January 2–3, D., E. L. This condition was undoubtedly due to dietary conditions, as the animal did not present a high temperature.

January 4, cow 3987 was inoculated with 150 cubic centimeters of mixed virulent blood from pig 237 and bull 3976. (For history of pig 237, see experiment 9.) (For history of bull 3976, see experiment 21.)

January 7, cow 3987 presented a morning temperature of 39.1° C. and an afternoon temperature of 40.4° C.

January 9, E. L.

January 10, E. L., bled to death for virulent blood to be used in immunization work.

In summing up the results of this experiment it will be noted that the potency of the virulent blood is not materially affected by mixing pig and cattle blood.

CATTLE DRENCHED WITH URINE FROM PIGS SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of cattle contracting rinderpest from the urine of pigs suffering with rinderpest.

EXPERIMENT 34

Cow 3978.—Known history prior to the experiment: Native Batanes bull, 2 years old, received at the laboratory and placed in quarantine October 23, 1915. It was kept under observation fifty-two days before it was used. During this period it did not present a high temperature or show any symptoms of sickness.

December 14, 1915, bull 3978 was drenched with 30 cubic centimeters of urine from pig 155. Water was added to the urine, making the quantity 250 cubic centimeters. This urine had also stood overnight.

History of pig 155 (see experiment 8):
  December 14: Temperature, a. m., 40.2° C.; p. m., 40.6° C.
  This animal ran a mild course of the disease and did not develop inappetence or diarrhœa.

Bull 3978 did not develop any reaction to this drench.

January 18, 1915, bull 3978 was inoculated with 400 cubic centimeters of virulent rinderpest blood procured from a carabao sick with rinderpest in Pampanga Province.

January 22, bull 3978 presented a rise in temperature.

January 24, this animal was bled to death for virulent blood to be used in immunization work.

EXPERIMENT 35

Bull 3993.—Known history prior to experiment: Native Batanes bull, 7 years old, received at the laboratory and placed in quarantine January 19, 1916, and kept under observation sixteen days before it was used in this experiment. At no time during this period did it have a high temperature or show any symptoms of disease.

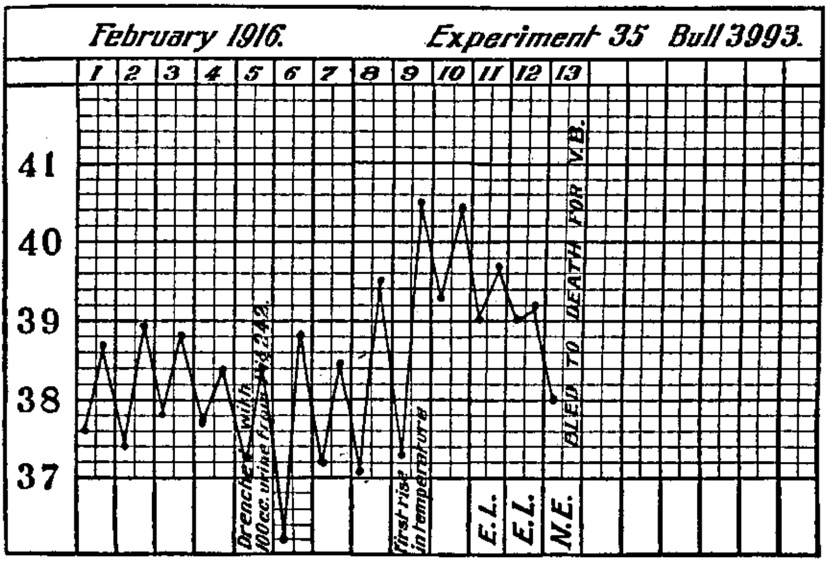

FIG. 7. Temperature chart of bull 3993 drenched with urine from pig.

February 5, 1916, bull 3993 was drenched with 100 cubic centimeters of fresh undiluted urine from pig 242.

History of pig 242 (see experiment 10):
- February 5: E. L.; a. m. and p. m. temperatures, 40.4° C.
- February 6: N. E.
- February 7: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

February 9, bull 3993 presented an afternoon temperature of 40.5° C.

February 12 and 13, E. L.

February 13, N. E., and was bled to death for virulent blood to be used in immunization work.

In summing up the results of these two experiments, with special reference to experiment 35, it will be noted that cattle can contract rinderpest by being drenched with urine from a pig sick with rinderpest. In experiment 34 there is a possibility that the urine was kept too long before being used, as has been demonstrated in previous work.(5)

CARABAOS EXPOSED TO PIGS SICK WITH RINDERPEST

The following experiment was designed to furnish information regarding the possibility of carabaos contracting rinderpest by being placed in contact with pigs sick with rinderpest. This experiment was conducted in stall No. 2.

EXPERIMENT 36

Carabao 57.—Known history prior to the experiment: This animal was a native carabao from Aparri, 8 years old, received at the laboratory and placed in quarantine May 11, 1916, and kept under observation seventy-two days before it was used in this experiment.

June 17, 1916, carabao 57 was used in an experiment in which an investigation was being conducted by Dr. H. W. Wade, of the Bureau of Science, and myself for the purpose of producing a vaccine for rinderpest by attenuating the virus by means of desiccated virulent blood and tissue pulp. This investigation will be reported on at a later date.

Carabao 57 gave no reaction to the injections administered.

July 22, 1916, carabao 57 was exposed to pigs 299 and 301.

History of pig 299:
- July 17, 1916: Inoculated with 50 cubic centimeters of virulent blood from carabao 53.
- July 20: First rise in temperature.
- July 21–23: N. E.
- July 24: E. L.
- July 25–26: D., E. L.
- July 27: Died of rinderpest, presenting typical lesions of that disease upon autopsy.

History of pig 301:
- July 17, 1916: Inoculated with 50 cubic centimeters of virulent rinderpest blood from carabao 53.
- July 19: First rise in temperature.
- July 21–23: N. E.
- July 24–27: E. L.
- This animal gradually recovered.

July 29, carabao 57 presented a rise in temperature, registering 39.3° C. in the morning.

July 31, D., N. E.

August 1–2, D., N. E.

August 3, died of rinderpest, presenting typical lesions of that disease upon autopsy.

In summing up the results of this experiment, it is proved that a carabao can contract rinderpest from pigs sick with that disease when the carabao is exposed to the pigs by direct contact.

CARABAOS INOCULATED WITH BLOOD FROM PIGS SICK WITH RINDERPEST

The following experiment was designed to furnish information regarding the possibility of carabaos contracting rinderpest from pigs when inoculated with blood from pigs sick with rinderpest.

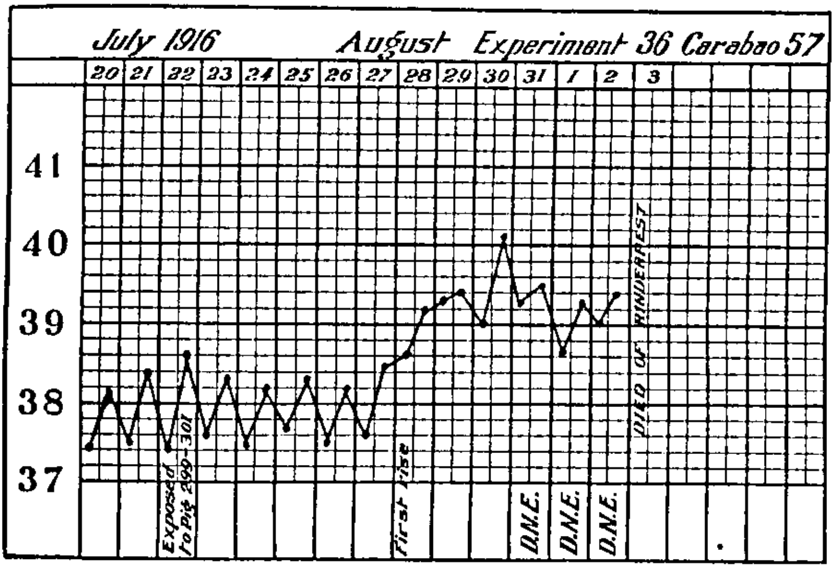

FIG. 8. Temperature chart of carabao 57 exposed to pig.

EXPERIMENT 37

Carabao 51.—Known history prior to the experiment: This animal was a native carabao from Aparri, 6 years old; it was received at the laboratory and placed in quarantine May 11, 1916, and was kept under observation thirty-three days before it was used in this experiment.

May 26, 1916, carabao 51 was used in an experiment on vaccination against rinderpest by the injection of desiccated virulent blood, but gave no reaction.

At no time during the period of observation did this animal present a high temperature or show any symptoms of sickness.

June 13, 1916, carabao 51 was inoculated with 10 cubic centimeters of blood from pig 300.

History of pig 300:

June 4, 1916: Inoculated with 10 cubic centimeters of virulent rinderpest blood procured from a carabao in Angono, Rizal Province.

June 9: First rise in temperature, registering, a. m., 39.2° C.; p. m., 40° C.

June 13: E. L.; bled to death, at which time the blood was procured to inject carabao 51.

June 19, carabao 51 presented a rise in temperature, registering, in the morning, 39.8° C.; in the afternoon, 40.7° C.

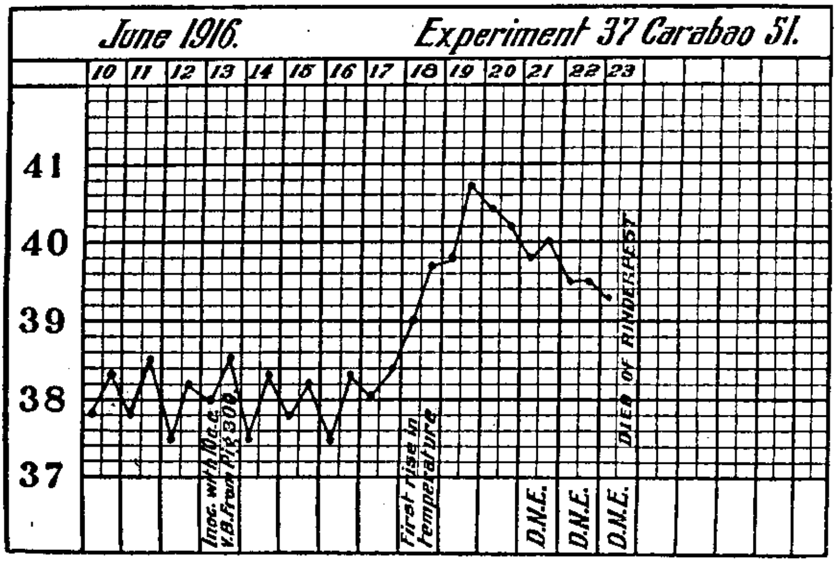

FIG. 9. Temperature chart of carabao 51 inoculated with blood from pig.

June 21–23 D., N. E.

June 23, died of rinderpest, presenting good lesions of that disease upon autopsy.

In summing up the results of this experiment, it will be noted that carabaos will contract rinderpest when injected with blood from pigs sick with rinderpest.

PIGS INOCULATED WITH BLOOD FROM CARABAOS SICK WITH RINDERPEST

The following experiments were designed to furnish information regarding the possibility of pigs contracting rinderpest when inoculated with virulent rinderpest blood from a carabao suffering with that disease.

EXPERIMENT 38

Pig 299.—Known history prior to the experiment: Native pig, 8 months old, purchased in Cavite Province in a locality which was free from rinderpest; it was brought to the laboratory and placed in quarantine May 30, 1916, and was kept under observation forty-eight days before it was used in this experiment.

June 2, E. L. This condition undoubtedly was caused by change in feed, as the animal did not present a high temperature or show any other symptoms of sickness during this period.

July 17, 1916, pig 299 was inoculated with 50 cubic centimeters of virulent rinderpest blood from carabao 53.

History of carabao 53:

July 1, 1916: Inoculated with 1.2 grams of dried virulent blood from carabao 54. This blood was dried in twenty hours and then kept in the ice box in a sealed tube for twenty-two hours.
June 10: First rise in temperature.
June 15: D., E. L.
June 16–19: D., N. E.
June 20: Died of rinderpest, presenting good lesions of that disease upon autopsy.

July 20, pig 299 developed a rise in temperature, registering, in the morning, 39.2° C.; in the afternoon, 40.2° C.

July 21–23, N. E.

July 24, E. L.

July 25–26, D., E. L.

July 27, died of rinderpest, presenting good lesions of that disease upon autopsy.

EXPERIMENT 39

Pig 301.—Known history prior to the experiment: Native pig, 7 months old, purchased in Cavite Province, brought to the laboratory and placed in quarantine May 30, 1916, and kept under observation forty-eight days before it was used in this experiment.

June 2, 3, and 4, this animal suffered from dietary disturbances, but immediately returned to normal. At no other time during this period did this animal present any symptoms of sickness.

July 17, 1916, pig 301 was inoculated with 50 cubic centimeters of virulent rinderpest blood from carabao 53. (For history of carabao 53 see experiment 38.)

July 19, pig 301 developed a rise in temperature, registering, in the afternoon, 40.3° C.

July 21–23, N. E.
July 24–27, E. L.

This animal gradually recovered.

In summing up the results of these two experiments, it will be noted that pigs will contract rinderpest when inoculated with virulent rinderpest blood from a carabao.

RINDERPEST TRANSMITTED FROM CATTLE TO PIGS BY MEANS OF THE CARETAKER

This experiment was designed to furnish information regarding the possibility of transmitting rinderpest from cattle sick with rinderpest to pigs by means of the caretaker. The sick cattle and the susceptible pigs were cared for by the same man.

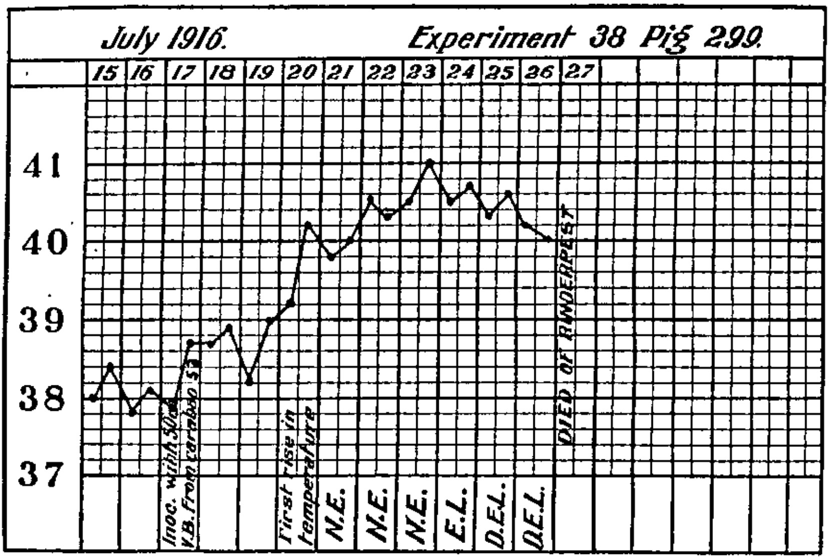

FIG. 10. Temperature chart of pig 299 inoculated with blood from carabao.

EXPERIMENT 40

On March 13, 1916, pigs 270, 271, 269, and 273 were placed in separate stalls in the same shed, where cattle sick with rinderpest were kept. These pigs were not kept in quarantine previous to their exposure and did not come in direct contact with the sick cattle, but were cared for by the same man, who took care of the sick cattle.

March 21, 1916, pig 270, native, three months old, presented a rise in temperature, registering, in the afternoon, 39.6° C.; this was eight days after the initial exposure.

March 26, D.

March 27–28, D., E. L.

March 28, died of rinderpest, presenting good lesions of that disease upon autopsy.

Pig 271, native, 5 months old, presented a rise of temperature March 28, 1916; this was fifteen days after the initial exposure. Its temperature on this day registered 40.4° C. in the afternoon.

April 3–5, N. E.

April 6, E. L.

April 7–8, N. E.

April 9, died of rinderpest, presenting good lesions of that disease upon autopsy.

Pig 269, native, 3 months old, presented a rise of temperature March 28, 1916; this was fifteen days after the initial exposure. Its temperature on this day registered 40.3° C. in the afternoon.

March 31, D.; morning temperature, 40.4° C.; afternoon temperature, 41.6° C.; this was the highest temperature recorded during the course of the disease.

April 1, 1916, D.

April 2, found dead in the morning. Presented good lesions of rinderpest upon autopsy.

Pig 273, native, 7 months old, presented a rise in temperature March 28, 1916, registering, in the afternoon, 40.1° C.; this was fifteen days after the initial exposure.

April 3–5, E. L.

April 6–7, D., E. L.

April 8–9, D., N. E.

April 10, D.

This animal gradually recovered.

In summing up the results of the exposure of these four pigs, it will be noted that pigs can contract rinderpest from cattle sick with that disease by means of the caretaker.

PIGS RECOVERED FROM RINDERPEST, AND THIS IMMUNITY TESTED TOWARD THAT DISEASE

The following experiment was designed to test the immunity of pigs to rinderpest after they had recovered from that disease. These animals were kept in the shed where the animals sick with rinderpest were placed, which gave them frequent exposures to animals sick with rinderpest in all stages of the disease.

EXPERIMENT 41

In this experiment pigs 94, 266, 267, 289, 291, 297, 301, 302, 155, and 154 were used.

Pig 94.—This animal was a Berkshire sow and had been hyperimmunized to hog-cholera virus and was later exposed to rinderpest by contact. It contracted and ran a mild course of the disease, recovering on December 5, 1914.

This animal was again exposed to rinderpest January 23, 1915, and these exposures have been continued up to date, covering a period of six hundred sixty-five days. During this time the pig has not shown any ill effects from these exposures, there having been no temperature or physical reaction.

Pig 266.—Recovered from rinderpest March 24, 1916. This animal was kept in the shed one hundred sixty days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 267.—Recovered from rinderpest April 2, 1916. This animal was kept in the shed one hundred fifty-one days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 289.—Recovered from rinderpest May 20, 1916. This animal was kept in the shed one hundred three days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 291.—Recovered from rinderpest May 13, 1916. This animal was kept in the shed one hundred ten days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 297.—Recovered from rinderpest June 28, 1916. This animal was kept in the shed sixty-four days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 301.—Recovered from rinderpest July 28, 1916. This animal was kept in the shed thirty-three days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 302.—Recovered from rinderpest June 8, 1916. This animal was kept in the shed eighty-four days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 155.—Recovered from rinderpest December 21, 1915. This animal was kept in the shed two hundred fifty-four days with animals sick with rinderpest and did not develop any ill effects from these exposures.

Pig 154.—Inoculated with 8 cubic centimeters of virulent rinderpest blood from bull 3995 on February 22, 1916. This animal did not react and was kept in the shed one hundred ninety-one days with animals sick with rinderpest and did not develop any ill effects from these exposures.

In summing up the results of the exposures of the ten preceding pigs, it will be noted that there is apparently a permanent immunity conferred on them against rinderpest after recovering from that disease, or at least for practically two years, which is the average life of a hog used for food purposes.

PIGS RECOVERING FROM RINDERPEST AND LATER BECOMING INFECTED WITH HOG CHOLERA

The following experiment was designed to furnish information as to whether pigs that had recovered from rinderpest were susceptible to hog cholera.

EXPERIMENT 42

On May 15, 1916, pigs 293, 294, 295, and 296 were received at the laboratory and placed in quarantine.

May 17, these four animals developed symptoms which were later identified as those of hog cholera. On this date the four animals were transferred to shed No. 3 and placed in the same pen with pigs 265, 272, 273, and 292, which had recovered from rinderpest.

Pig 265 recovered from rinderpest March 22, 1916.

May 17, 1916, this animal was exposed to the four pigs sick with hog cholera; this was fifty-six days after it had recovered from rinderpest.

May 30, pig 265 presented a rise in temperature.

June 7–11, E. L.

June 12, died of hog cholera, presenting good lesions of that disease upon autopsy.

Pig 272 recovered from rinderpest April 10, 1916.

May 17, 1916, this animal was exposed to the four pigs sick with hog cholera; this was thirty-seven days after it had recovered from rinderpest.

May 30, pig 272 presented a rise in temperature.

June 10–16, E. L.

June 17–23, D., E. L.

June 23, died of hog cholera, presenting good lesions of that disease upon autopsy.

Pig 273 recovered from rinderpest April 11, 1916.

May 17, 1916, this animal was exposed to the four pigs sick with hog cholera; this was thirty-six days after it had recovered from rinderpest.

June 1, 1916, pig 273 presented a rise in temperature.

June 10–15, E. L.

June 16–19, N. E.

June 20, D., E. L.

June 21–23, E. L.

June 23, this animal was killed and upon autopsy presented good lesions of hog cholera.

Pig 292 recovered from rinderpest May 9, 1916.

May 17, 1916, this animal was exposed to the four pigs sick with hog cholera; this was eight days after it had recovered from rinderpest.

May 30, 1916, pig 292 presented a rise in temperature. This animal ran a mild course, was killed on June 23, and presented a few lesions of hog cholera upon autopsy.

From the results obtained in the preceding experiment, it will be noted that pigs that have recovered from rinderpest are susceptible to and can die of hog cholera, which proves that there is no immunity toward hog cholera conferred upon a pig that has recovered from rinderpest.

PIGS HYPERIMMUNIZED TO HOG CHOLERA AND LATER CONTRACTING RINDERPEST

The following experiments were designed to ascertain whether pigs that had been hyperimmunized against hog cholera would contract rinderpest when exposed to cattle sick with rinderpest.

EXPERIMENT 43

Pig 1.—This animal received its last injection of 450 cubic centimeters of virulent hog-cholera blood on October 15, 1914.

November 16, 1914, pig 1 was exposed to bull 3845.

History of bull 3845:

November 9, 1914: Inoculated with 200 cubic centimeters of virulent blood from bull 3780.
November 11: Bull 3845 presented a rise in temperature.
November 11–16: D.
No. 17–18: D., N. E.
November 19: Died of rinderpest.

November 20, pig 1 presented a rise in temperature, registering, in the morning, 39.8° C.; in the afternoon, 41.9° C.

November 22–27, N. E.

November 28, E. L.

This animal's temperature gradually subsided to normal, and it made a complete recovery.

EXPERIMENT 44

Pig 2.—This animal received its last injection of 350 cubic centimeters of virulent hog-cholera blood on October 14, 1914.

November 9, 1914, pig 2 was exposed to bull 3780.

History of bull 3780:

November 21, 1914: Inoculated with 200 cubic centimeters of virulent rinderpest blood from bull 3838.

November 5: Bull 3780 presented a rise in temperature.

November 11: D., N. E.

November 12: E. L.

This animal gradually recovered.

November 13, pig 2 presented a rise in temperature, registering, in the morning, 40° C.; in the afternoon, 40.3° C.

November 16, afternoon temperature, 41.3° C.

The temperature of this animal was high until November 21, when it subsided to normal, and the animal made a prompt recovery.

EXPERIMENT 45

Pig 4.[2]—This animal received its last injection of 450 cubic centimeters of virulent hog-cholera blood on October 13, 1914.

December 1, 1914, pig 4 was exposed to bull 3847.

History of bull 3847:

November 28, 1914: Inoculated with 100 cubic centimeters of virulent rinderpest blood from bull 3873.

December 1: Bull 3847 presented a rise in temperature.

December 5–8: D., E. L.

December 9–10; D.

This animal gradually recovered.

December 8, pig 4 presented an afternoon temperature of 40.5° C.

December 9, the afternoon temperature was 41° C.

The temperature of this animal was high until December 15, when it subsided to normal, and the pig recovered.

EXPERIMENT 46

Pig 5.—This animal received its last injection of 400 cubic centimeters of virulent hog-cholera blood on October 14, 1914.

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[2] Pig 3 was not used in this series of experiments.

November 23, 1914, pig 5 was exposed to bull 3846.

History of bull 3846:

November 16, 1914: Inoculated with 100 cubic centimeters of virulent rinderpest blood from bulls 3716 and 3845.
November 19: Bull 3846 presented a rise in temperature.
November 21: D., E. L.
November 22–23: D.
November 24–25: D., N. E.
November 26, died of rinderpest.

November 25, pig 5 presented an afternoon temperature of 41.1° C.

November 26–27, the afternoon temperature was 41.7° C.

November 28, N. E.

November 29, D., N. E.

November 30, E. L.

December 1, E. L.

This animal's temperature returned to normal December 6, and the pig recovered.

EXPERIMENT 47

Pig 6.—This animal received its last injection of 300 cubic centimeters of virulent hog-cholera blood on October 14, 1914.

November 16, 1914, pig 6 was exposed to bull 3846. (For history of bull 3846, see experiment 46.)

November 19, pig 6 presented a morning temperature of 39.7° C.; and an afternoon temperature of 41.7° C.

November 23–27, N. E.

November 28–30, E. L.

December 1, E. L.; this animal was killed on this date.

EXPERIMENT 48

Pig 7.—This animal received its last injection of 450 cubic centimeters of virulent hog-cholera blood on October 15, 1914.

November 12, 1914, pig 7 was exposed to bull 3842.

History of bull 3842:

November 7, 1914: Inoculated with 100 cubic centimeters of virulent rinderpest blood from Pampanga Province.
November 13: Bull 3842 presented a rise in temperature.
This animal ran a rather mild form of the disease and recovered.

November 16, pig 7 presented a morning temperature of 39° C. and an afternoon temperature of 41.1° C.

November 17, the afternoon temperature was 41.2° C.

November 21, this animal's temperature had subsided to normal, and the pig made a rapid recovery.

In summing up the results of these experiments, it will be noted that pigs that have been hyperimmunized to hog cholera can contract rinderpest when exposed to cattle sick with rinderpest. These results prove that there is no immunity conferred against rinderpest by the hog-cholera virus, although the animals ran a milder course of the disease than has been usually witnessed in the laboratory. This condition may be considered from two viewpoints: First, that possibly the strain of rinderpest virus was not very virulent; and secondly, that the hyperimmunization against hog cholera may have increased the resistance toward rinderpest.

CONCLUSIONS

1. From the results obtained in experiments 1 to 10, inclusive, it is proved that pigs can contract rinderpest when exposed to cattle sick with rinderpest. The disease thus contracted by pigs may terminate in death, unthriftiness, or complete recovery.
2. From the results obtained in experiments 11 to 13, inclusive, it is proved that pigs can contract rinderpest when exposed to pigs sick with rinderpest.
3. From the results obtained in experiment 14 it is proved that a pig can contract rinderpest from pigs sick with rinderpest by means of the caretaker.
4. From the results obtained in experiments 15 and 16 it is proved that pigs can contract rinderpest when inoculated with blood drawn from pigs sick with rinderpest.
5. From the results obtained in experiments 17 to 20, inclusive, it is proved that pigs can contract rinderpest when inoculated with blood drawn from cattle sick with rinderpest.
6. From the results obtained in experiment 21 it is proved that a pig can contract rinderpest when inoculated with mixed blood drawn from cattle and pigs sick with rinderpest.
7. From the results obtained in experiment 22 it is proved that a pig can contract rinderpest when it is drenched with blood drawn from carabaos sick with rinderpest.
8. From the results obtained in experiments 23 to 28, inclusive, it is proved that cattle can contract rinderpest when exposed to pigs sick with rinderpest. However, cattle do not contract the disease very readily by this method, and the exact reason for this has not been ascertained at the present writing.
9. From the results obtained in experiments 29 to 32, inclusive, it is proved that cattle can contract rinderpest when inoculated with blood drawn from pigs sick with rinderpest.

10. From the results obtained in experiment 33 it is proved that cattle can contract rinderpest when inoculated with mixed blood drawn from pigs and cattle sick with rinderpest.

11. From the results obtained in experiments 34 and 35 it is proved that cattle can contract rinderpest when drenched with urine collected from pigs sick with rinderpest.

12. From the results obtained in experiment 36 it is proved that carabaos can contract rinderpest when exposed to pigs sick with rinderpest.

13. From the results obtained in experiment 37 it is proved that carabaos can contract rinderpest when inoculated with blood from pigs sick with rinderpest.

14. From the results obtained in experiments 38 and 39 it is proved that pigs can contract rinderpest when inoculated with blood from carabaos sick with rinderpest.

15. From the results obtained in experiment 40 it is proved that pigs can contract rinderpest from cattle sick with rinderpest by means of the caretaker.

16. From the results obtained in experiment 41 it is proved that pigs remain immune to rinderpest after their recovery from that disease for at least six hundred sixty-five days and undoubtedly during the rest of their existence.

17. From the results obtained in experiment 42 it is proved that pigs that have recovered from rinderpest are susceptible to and may die from hog cholera, which shows that there is no immunity against hog cholera conferred upon a pig that has passed through an attack of rinderpest.

18. From the results obtained in experiments 43 to 48, inclusive, it is proved that pigs that have been hyperimmunized to hog cholera are susceptible to rinderpest when exposed to cattle sick with rinderpest. Although the pigs used in these experiments did not run as severe a course of the disease as the average pig, there is a possibility that the strain of virus with which they were infected was not of the most virulent type. This condition is frequently noticed in rinderpest in cattle. One strain may cause a high mortality, while another may cause a comparatively low mortality. Thus it is rather difficult to state definitely that the hyperimmunization to hog cholera was of any benefit to the pigs when they were exposed to rinderpest.

19. In summing up the results of all of these experiments, it will be noted that cattle, carabaos, and pigs vary but slightly in susceptibility to rinderpest and that the disease can be transmitted practically as readily from one kind of animal to the other as among the individuals of a single species.

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ILLUSTRATIONS

[Plates lent by the Bureau of Agriculture.]

PLATE I

A group of pigs sick with rinderpest.

PLATE II

FIG. 1. A pig in the last stages of rinderpest.
2. A pig in the last stages of rinderpest, possessing marked eye lesions.

TEXT FIGURES

FIG. 1. Temperature chart of pig 240 exposed to bull.
2. Temperature chart of pig 212 exposed to pig.
3. Temperature chart of pig 219 inoculated with blood from pig.
4. Temperature chart of pig 241 inoculated with blood from bull.
5. Temperature chart of cow 3995 exposed to pig.
6. Temperature chart of bull 3839 inoculated with blood from pig.
7. Temperature chart of bull 3993 drenched with urine from pig.
8. Temperature chart of carabao 57 exposed to pig.
9. Temperature chart of carabao 51 inoculated with blood from pig.
10. Temperature chart of pig 299 inoculated with blood from carabao.

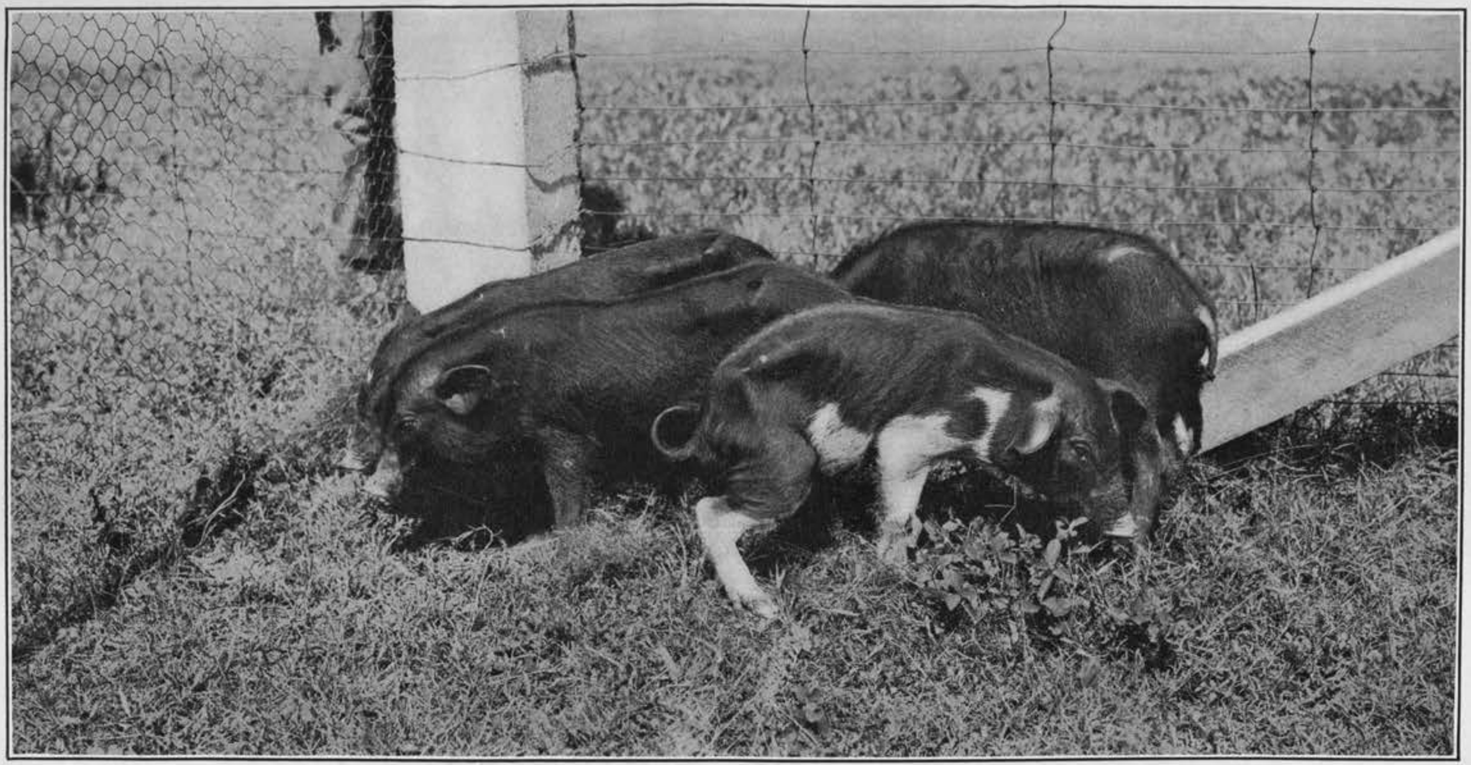

PLATE I. A GROUP OF PIGS SICK WITH RINDERPEST.

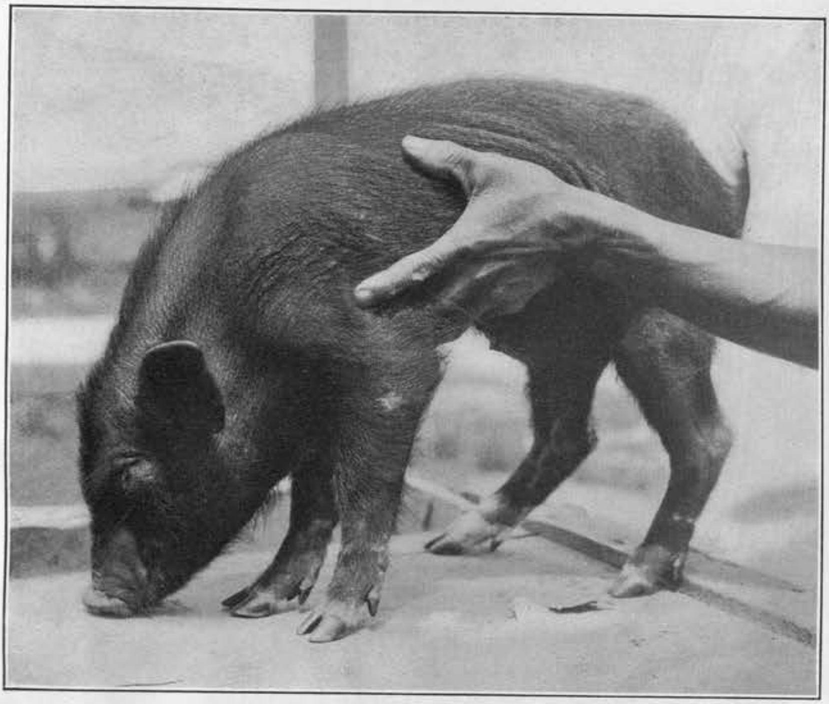

Fig. 1. A pig in the last stages of rinderpest.

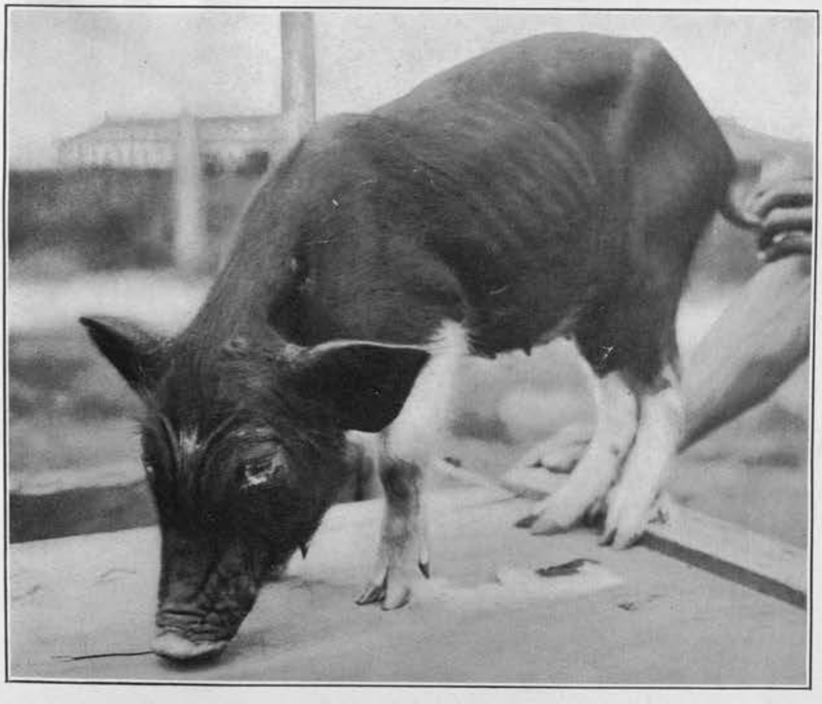

Fig. 2. A pig in the last stages of rinderpest, possessing marked eye lesions.

PLATE II.